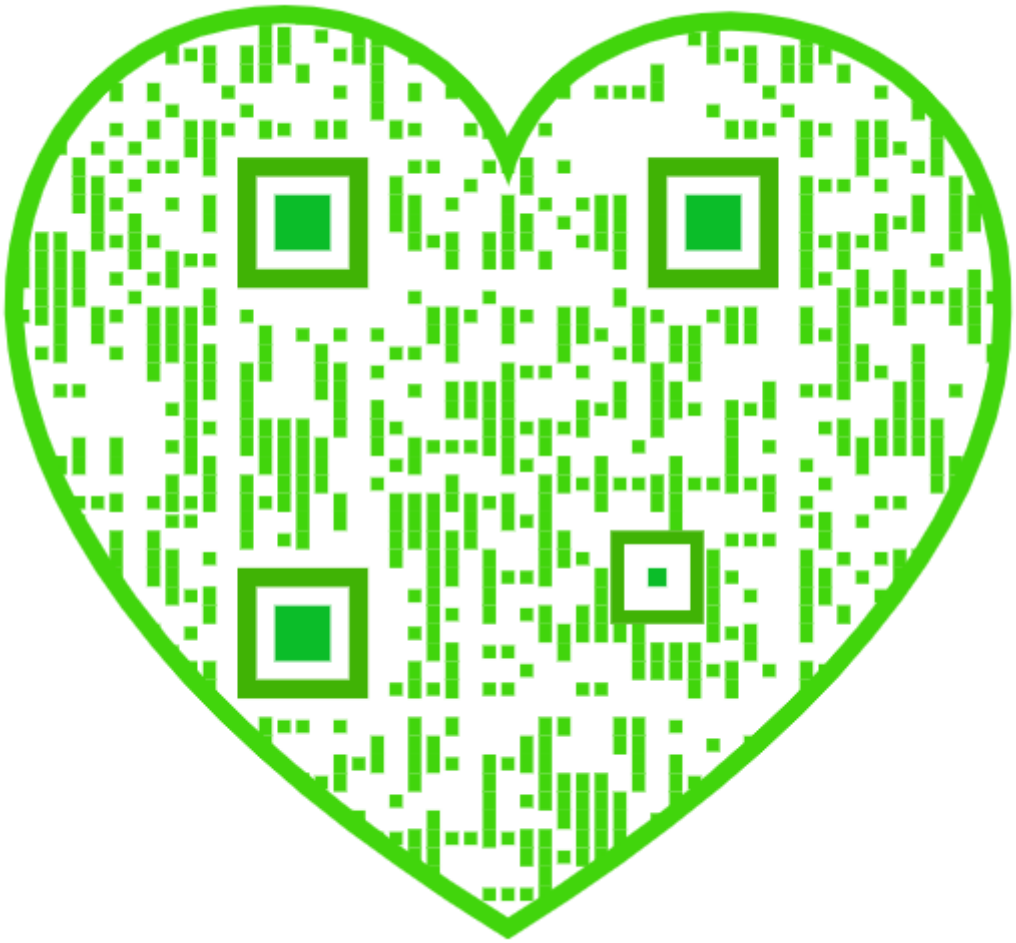

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RHINOLOGY

LARYNGOLOGY AND OTOLOGY

AND THEIR

SIGNIFICANCE IN GENERAL MEDICINE

BY

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PRIVATDOCENT AT THE UNIVERSITY OF LEIPZIG

Authorized Translation from the German

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CONSULTING SURGEON TO THE NEW YORK NOSE AND THROAT HOSPITAL
AND TO THE DIPHTHERIA AND SCARLET FEVER HOSPITALS

PHILADELPHIA AND LONDON

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EDITOR'S PREFACE.

At the present time, when it is the fashion for almost every specialist to pad his individual work and announce a book on the ear, nose, and throat,—which upon perusal is generally found to cover ground already occupied,—it is certainly with pardonable enthusiasm that we greet a masterly treatise of a thoroughly original type, the intrinsic worth of which warrants its appearance in our own language. Friedrich has realized that the general practitioner must acquaint himself with the rapid advances in the modern teaching of otolaryngology, and he has constructed a book so rich in statistics and reference, so learned in its argumentative deductions, and at the same time so convincing in the manner of conservative presentation, that no specialist can afford to neglect the opportunity of acquainting himself with the subject-matter of his work. The results of the vast clinical experience of the author, the detailed reports, and the extensive bibliography make the volume valuable alike to the specialist and the general practioner.

Far too little attention has been paid in the past to early symptomatic manifestations in the respiratory tract; nor has sufficient study been given to the reflex neuroses of the ear and air-passages and their diagnostic significance.

One can not read the book without admiration for a physician who is able at once to be observant of the minutest detail of constitutional disturbance, and also to possess so intimate a knowledge of the specialties whose reciprocal relations he so ably defines.

The chapters treating of nervous diseases are most interesting and rich in new material, as are also those upon the exanthemata and their sequels.

In the translation there has been no effort made to render into elegant English the characteristic construction of the German text. To preserve the exact meaning of the

author and his individual style of expression has been our aim, and the work is reproduced from a strictly scientific point of view.

The attention which has been given to the morbid anatomy and pathology is exceptional, and the book as a whole ranks among the most progressive works of to-day.

The editor has no hesitation in indorsing the book as the best treatise upon the relationship of general diseases to those of the ear, nose, and throat that has appeared up to this time.

H. Holbrook Curtis.

118 Madison Avenue, New York City.

PREFACE.

In these days of specialism there is a laudable tendency to tighten the bonds that unite the daughter to the mother science. On every hand we see the publication of works destined to show the correlation between various branches of medicine, and to awaken the interest of representatives of the various specialties for one another's work by defining the lines where their respective provinces meet.

The present book belongs to this category, and the author's object has been to point out the interdependence between diseases of the entire organism and diseases of the nose, pharynx, larynx, and ears.

It is a somewhat hazardous experiment, this attempt to unite the three specialties rhinology, laryngology, and otology in a single treatise, and I am well aware of the opposition it is destined to meet from the extreme advocates of specialism. But let us examine the relation between these three specialties. A study of the history shows us that rhinology was added to laryngology in response to a practical demand, and that recent developments have shown that otology stands more and more in need of rhinology—so much so that a distinction might be made between rhino-laryngology and rhino-otology. But we ought rather to oppose this subdivision, since the study of rhinology is as important for the successful practice of laryngology, as a knowledge of nasal affections is indispensable in the practice of otology; in other words, one specialty encroaches on the domain of another, and it is impossible to establish definite boundaries.

Whenever it becomes necessary to examine more carefully certain regions of the body, anatomically and physiologically distinct, the natural result is the growth of a specialty.

As experience shows that certain portions of the organism

which, owing to their position and function, can not be reached by ordinary methods of examination practised in general medicine, demand the development of special methods to enable us to examine new regions both in the healthy and in the diseased state, and as with the growth of our knowledge additional facts are discovered, the field gradually widens, and new departments spring into existence, which are only too often regarded by both physicians and laymen as isolated domains of the general science.

There must, of course, be a period in the development of a specialty during which those who practise it devote their entire energy to the study of the anatomy and physiology of the new regions, and to the creation of a special pathology and method of treatment adapted to the peculiarities of the parts concerned. Once this foundation is established, however, it becomes important to incorporate the new discoveries in the scheme of general medicine. A specialty should not be regarded as a thing apart and a kind of appendage; it should take an active part in all the problems with the solution of which general medicine is concerned. To do this an active cooperation between general medicine and every one of the various specialties is indispensable. Whenever it is lacking, the specialty is in danger of becoming a mere source of revenue and of losing its scientific significance; while, on the other hand, the general practitioner will fail to recognize special symptoms which might have been of the greatest importance to him in the recognition and treatment of his cases.

In taking this standpoint and in describing the relations which manifest themselves as disturbances of the general organism in disease of special parts, or as disturbances of special parts in general disease, I lay no claim to originality, for several works on our specialties have appeared with a special reference to general medicine. Moritz-Schmidt, in his excellent book on "Diseases of the Upper Air-passages," has followed the same lines, "writing from practice for practice"; Löri discusses "Alterations of the Pharynx, Larynx, and Trachea due to Other Diseases"; while Moos and Haug discuss "The Diseases of the Ear in their Relation to General Diseases," one in his chapter on the Etiology of the Diseases of the Ear in Schwartze's "Handbuch der Otologie," the other in a special monograph.

My undertaking a new work on this theme merely shows the abundance of material accumulated during the last few years, and the development of new points of view that justify the publication of another book on the subject. It has been my endeavor to confine myself to the positive, and, disregarding speculation, to present to the reader nothing but exact and well-established information.

The treatment of the subject is purposely succinct, especially the description of the commoner diseases. A detailed description of special symptomatology is not within the scope of this book, which does not pretend to be a special text-book in the ordinary sense, being intended to awaken the interests of both the general practitioner and the specialist in certain matters which appear to me to demand special attention and further elaboration. As I have drawn freely from the entire literature, bibliographic notes seemed to me indispensable. They do not pretend to anything like completeness, but I have, I hope, cited the most important works, a reference to which will enable the reader to elucidate any doubtful questions that may present themselves.

E. P. Friedrich.

CONTENTS.

I. DISEASES OF THE RESPIRATORY ORGANS.

PAGE

1. General Remarks on the Relations Existing between the Nose, Pharynx, and Larynx 17
2. Relations Existing between the Nose, Pharynx, Larynx, and Lungs . 19
   - Significance of the Upper Air-passages in the Physiology of Breathing 19
   - Diseases of the Lungs Due to Disturbances of the Physiologic Function of the Upper Air-passages 26
   - Diseases of the Lungs in Morbid Conditions of the Upper Air-passages 28
   - Alterations in the Upper Air-passages in Diseases of the Lungs . 31
   - Alterations in the Upper Air-passages in Diseases of the Mediastinum 33
3. Relations between the Upper Air-passages and the Ears 35
   - The Effect of Disturbances of the Normal Function of the Eustachian Tube 36
   - Disturbances of the Function of the Eustachian Tube Due to Alterations in the Upper Air-passages 40
   - Diseases of the Middle Ear Due to Infection from the Post-nasal Space 43
4. The Effect of Various Diseases of the Respiratory Organs on the Ears 47

II. DISEASES OF THE CIRCULATORY SYSTEM.

1. Diseases of the Heart and Blood-vessels in Their Relation to the Nose, Pharnyx, and Larynx 52
2. Diseases of the Heart and Blood-vessels in Their Relation to the Ear 59

III. DISEASES OF THE DIGESTIVE SYSTEM.

1. Diseases of the Digestive System in Their Relation to the Upper Air-passages 68
   - Diseases and Changes in Form of the Oral Cavity in Disturbances of Nasal Respiration 68
   - Diseases of the Digestive Organs in Relation to the Nose, Throat, and Larynx 70
2. Digestive System and Diseases of the Ear 76

IV. DISEASES OF THE BLOOD.

PAGE
1. Anemia 81
2. Leukemia 83
Alterations in the Upper Air-passages in Leukemia 83
The Manifestations of Leukemia in the Ear 86
3. Hemorrhagic Diatheses 89

V. CHRONIC CONSTITUTIONAL DISEASES.

1. Rachitis 92
2. Acromegaly 95
3. Diabetes Mellitus 96
4. Gout 102
Ictus Laryngis Occurring in the Course of Obesity, Gout, and Diabetes 104

VI. ACUTE INFECTIOUS DISEASES.

1. Measles 108
2. Scarlatina 112
3. Varicella 120
4. Variola 121
5. Typhoid Fever 123
6. Influenza 132
Aural Complications in Influenza 134
7. Parotitis Epidemica (Mumps) 138
8. Acute Rheumatoid Arthritis 139
9. Diphtheria 142
10. Erysipelas 147
11. Malaria 148

VII. CHRONIC INFECTIOUS DISEASES.

1. Tuberculosis and Lupus 151
Tuberculosis of the Nose 158
Tuberculosis of the Pharynx 161
Tuberculosis of the Larynx 162
Tuberculosis of the Ear 169
Lupus 174
2. Leprosy 177
3. Malleus Humidus (Glanders) 183
4. Foot-and-mouth Disease 184
5. Anthrax 185
6. Actinomycosis 185
7. Rabies 186
8. Trichinosis 187

VIII. DISEASES OF THE KIDNEY.

Edema of the Pharynx and Larynx 188
Hemorrhages in the Pharynx and Larynx 189
Nephritic Aural Diseases 189

IX. DISEASES OF THE SKIN AND OF THE SEXUAL ORGANS.

PAGE
1. Diseases of the Skin 193
2. The Influence of Normal or Pathologically Altered Sexual Functions on the Upper Air-passages 197
Relation of the Sexual Organs to the Upper Air-passages . . . 197
Relations between the Sexual Organs and the Ears 201
3. Gonorrhea 203
4. Syphilis 205

X. DISEASES OF THE EYE.

1. Relations between the Eye and the Nose 224
2. Relations between the Eyes and the Ears 234

XI. INTOXICATIONS.

Acids and Alkalies 242
Iodids 243
Arsenic and Lead 245
Mercury 246
Copper, Phosphorus, etc. 247
Quinin, Salicylic Acid, etc. 248
Chloroform, Tobacco, Alcohol 249

XII. NERVOUS DISEASES.

1. General Remarks on Diseases of the Larynx in Diseases of the Central Nervous System 251
Diseases of the Sensory and Motor Nerves of the Larynx . . . 251
Localization of Centers for Movement of the Vocal Cords in the Central Nervous System, and the Effect of Diseases of the Central Nervous System 258
2. General Remarks on the Aural Disturbances Produced in Diseases of the Central Nervous System 263
The Mechanism of Functional Disturbances in the Ear and the Electric Reactions of the Auditory Nerve 263
The Localization of the Ear in the Central Nervous Organs . . 270
3. Nervous Diseases which Produce Definite Alterations in the Nose, Pharynx, and Larynx, and in the Ears 275
Diseases of the Spinal Cord 275
Tabes Dorsalis 275
Multiple Sclerosis 289
Diseases of the Medulla Oblongata 291
Syringomyelia 291
Progressive Amyotrophic Bulbar Paralysis 292
Neuroses 293
Paralysis Agitans 293
Epilepsy 294
Chorea Minor 295
Hysteria 295

APPENDIX.

PAGE

Nasal Reflex Neuroses 306
The Significance of Some of the Cranial Nerves in Rhinology and Otology, 314
The Trifacial Nerve 314
The Chorda Tympani 320
The Facial Nerve 323
Diseases of the Meninges and of the Cerebral Sinuses 325
Their Significance in Connection with the Nose, Larynx, and Ears . . 325
Diseases of the Meninges in Nasal Affections 328
Diseases of the Meninges and of the Cerebral Sinuses in Ear Disease 330

INDEX 337

RHINOLOGY

LARYNGOLOGY AND OTOLOGY

AND THEIR

SIGNIFICANCE IN GENERAL MEDICINE

I. DISEASES OF THE RESPIRATORY ORGANS.

1. GENERAL REMARKS ON THE RELATIONS EXISTING BETWEEN THE NOSE, PHARYNX, AND LARYNX.

The upper air-passages, comprising the nose, pharynx, and larynx, present a canal of varying form and diameter, lined in its entire extent, except where the respiratory and alimentary tracts cross each other in the pharynx, by mucous membrane covered with ciliated columnar epithelium; so that nose, pharynx, and larynx imperceptibly merge one into the other without the interposition of a sharp line of demarcation. It follows that pathologic changes in any portion of the upper air-passages are not sharply limited in their local effects and ultimate consequences, but invade adjacent areas quite irrespective of the anatomic boundaries of nose, pharynx, and larynx.

It is well known that catarrhal affections of the upper air-passages are not limited to a circumscribed area; they display, on the contrary, a peculiar descending character, as it is called, beginning in the nose as an acute rhinitis and invading at certain definite intervals the pharynx and the larynx.

The comparative immunity enjoyed by the larynx as compared to the pharynx is not altogether accidental, although to a certain extent dependent on accidental causes, for it is generally admitted that progressive morbid processes meet with a certain resistance wherever the character of the epithelium changes, which resistance may be sufficient to arrest their further advance. Now, the ciliated columnar epithelium of the nose and nasopharynx is replaced in the oral pharynx by squamous epithelium, which extends as far as the upper border of the larynx; but at this point the epithelium returns to the ciliated columnar type of the higher air-passages, and this type is retained

throughout the interior of the larynx—with the exception of a zone of squamous epithelium extending over the interarytenoid notch to the posterior wall and to the vocal cords. Hence we can readily understand that the boundaries between these various kinds of epithelium oppose to the progress of an acute catarrh a barrier more or less insurmountable, according to the intensity of the process and the disposition of the individual. In the comparatively rare cases where the larynx becomes involved in a descending catarrh, the laryngeal symptoms develop several days after the first appearance of the nasal and pharyngeal symptoms, or even after convalescence has begun in the higher air-passages.

Ascending catarrh, on the contrary, differs diametrically from the descending form in the matter of frequency, and it seldom or never happens that an acute pharyngitis or laryngitis spreads to the higher portions of the respiratory tract.

With the infectious diseases, especially diphtheria, the case is quite different; they may originate either in the pharynx or in the nose, although, as a matter of fact, they usually appear first in the pharynx, and spread from that region either upward into the nose or downward to the larynx.

The relation between the nose and the nasopharynx is a particularly intimate one, so much so that only very few diseases are limited to one or the other of these two structures. Any chronic condition leading to obstruction and to the passage of morbid products—such as mucus and pus—into the nasopharynx exerts an injurious effect on that structure; and, conversely, any disease of the nasopharynx, by causing chronic enlargement of the pharyngeal tonsils, thereby obstructing the nasal passages and interfering with nasal respiration, sets up a congestive catarrh; the secretions accumulate as the expiratory blast is no longer able to remove them, and a hyperplasia of the mucous membrane eventually results.

If, on account of obstruction, the nasal secretion is unable to make its escape outward and flows back into the nasopharynx, the harm which results is not confined to this locality; the discharges trickling down along the posterior pharyngeal wall accumulate in the oral pharynx, and the subsequent course of the disease then depends on the

quantity and consistency of the morbid secretion. If the patient is unable to remove it by hawking and coughing, it will adhere to the posterior pharyngeal wall in the form of a thick, tenacious coating, and thence will gradually spread to the posterior wall of the larynx. This mode of spreading from the nose to the pharynx and larynx is especially characteristic of certain definite diseases, the most typical of which is atrophic rhinitis with fetor and crust formation. The greenish-yellow, foul-smelling crusts with which the atrophied nasal cavities are covered—as the walls of a room are covered with wall-paper—are also found clinging to the roof and posterior wall of the pharynx, while in the larynx a tenacious material accumulates on those parts which are least concerned in the movements of phonation and respiration, especially in the region below the glottis. Similar consequences attend any chronic inflammation of the nose, accompanied with copious secretion and suppurative processes in the tributary cavities of the nose, whenever the position of their openings permits a backward flow of pus.

2. RELATIONS EXISTING BETWEEN THE NOSE, PHARYNX, LARYNX, AND LUNGS.

SIGNIFICANCE OF THE UPPER AIR-PASSAGES IN THE PHYSIOLOGY OF BREATHING.

Leaving the description of the interdependence of nose, pharynx, and larynx, which really belongs to the domain of special pathology, we now turn our attention to the influence exerted on the lungs by disease of the upper air-passages.

The first requisite for a thorough understanding of this subject is a knowledge of the physiologic significance of the air-passages in the act of respiration. They should not be viewed merely in the light of canals for the transmission of the inspired air; for each segment has a special function of its own and contributes to the preparation of the air for reception in the lungs, and this function can not remain in abeyance without detriment to the organism.

We begin with the consideration of the upper air-passages as the respiratory pathway and with the changes experienced by the inspiratory air-current during its passage through the nose.

I. The Nose as the Respiratory Pathway.—The nose is the portal through which the air gains admittance to the body, and it has certain special functions to perform which lend to it a greater importance than belongs to the pharynx and larynx. It is charged with the duty of preparing the air for its entrance into the deeper air-passages, in the following ways:

1. By removing foreign substances as much as possible.
2. By warming the air.
3. By imparting to the air the requisite degree of moisture.
4. A subordinate function consists in protection of the organism by means of the sense of smell and the nasal reflexes.

In order to gain a full understanding of these various functions let us examine the path followed by the air in its transit through the nose. Even at the present day we frequently hear of the division of the nasal cavity into a respiratory and an olfactory region, the former corresponding with the maxillary, and the latter with the ethmoidal, portion. It should follow from this subdivision that the lower half of the nose, as far as the middle turbinated bone, is concerned exclusively in the act of breathing, while the remaining upper half subserves solely the sense of smell. Experimental researches prove, however, that such a division is not justified, either by the nature of the respiratory air-current or by the distribution of the nerves, to which we shall return later. Experiments have been made by Paulson, Kayser,[1] Franke,[2] and Scheff,[3] and those of Scheff have recently been repeated and confirmed by Danziger.[4] The perfect agreement of these experiments and the convincing care with which they were performed, justify us in rejecting the older theory, according to which the respiratory air-current passes only through the lowest segment of the meatus nasi communis: that is, the space between the inferior turbinated bone and the septum. The most important fact brought out by recent investigations is that the respiratory current passes principally through the middle and upper parts of the nose, and hardly touches the

[1] "Zeitschr. f. Ohr.," vol. XX, p. 96.
[2] "Arch. f. Laryng.," vol. I, p. 230.
[3] "Klin. Zeit- u. Streitfr.," Vienna, 1895, vol. IX, part II.
[4] "Mon. f. Ohr.," 1896, p. 331.

inferior meatus under normal conditions. As the entrance to the nose is in the horizontal plane, the current of air, on entering, rises in a line parallel to the dorsum of the nose; it is then deflected backward in the region of the agger nasi (which in man is rudimentary), describing a curve, the concavity of which faces downward, while its apogee may project above the superior, and never falls below the middle turbinated bone, and passes out through the upper half of the posterior nares. According to Franke, the air-current also forms an eddy somewhere in the region of the inferior turbinated bone. During expiration the curve is flattened, its elevation being in direct proportion to the depth of the inspiratory movement.

While we must accept this as the type of nasal respiration to be considered in judging of pathologic conditions, we must also take into account the shape of the external nose, the position of the anterior nares, and the width from side to side of the internal openings—which depends on the prominence of the plica alaris. The significance of these factors was shown by Kayser in his experiments to determine the manner of aspiration in variously shaped noses. In the narrow, aquiline variety of nose, in which the external opening is horizontal and the inner opening usually small, the air-current follows the direction which has been described; on the other hand, we have the testimony of various authors that in the broad, turned-up, so-called pug-nose, in which the space between the septum and the plica vestibuli is large, the air-current enters in a more horizontal direction, and is directed toward the lower portion of the nose.

This apparently complicated arrangement enables the nose to fulfil the three different functions which pertain to it in the preparation of the respiratory air-current, by insuring the greatest possible extent of contact with the walls of the nasal cavities.

1. Removal of Foreign Substances from the Respiratory Air-current.—When we consider the great variety of conditions with which we are surrounded, it is self-evident that the purity of the air, which depends on the presence or absence of dust and gaseous substance, is subject to considerable change. Under the head of dust we have to consider solid particles of a mineral or vegetable nature and microorganisms.

The nose is provided with various means of defense against the entrance of these deleterious substances: the vibrissæ which line the inner margin of the nostrils, the moist surface of the mucous membrane, the reflex act of sneezing, and, lastly, a bactericidal property which probably resides in the mucous secretion.

The vibrissæ act like a coarse filter which arrests the larger particles. The moist surface of the mucous membrane attracts and holds fast any foreign substances in the air-current as it passes through the narrow and complicated passages of the nose. This occurs especially at certain points where the current impinges on the surface of the mucous membrane and is deflected, necessitating a certain amount of friction between the air and the walls of the cavities. In consequence of this friction, the dust particles suspended in the air-current are brought into close contact with the mucous membrane, and stick fast to its moist surface, later to be swept out by the outward current of the ciliated epithelium. One important region of this kind is found on the cartilage of the septum, at the level of the inferior turbinated bone, at the spot where the inspired current, after being deflected inward by the plica vestibularis, strikes the septum; another corresponds to the posterior pharyngeal wall, opposite the posterior nares. The purification which the air undergoes in the nose does not, however, entirely prevent the inhalation of dust into the lungs, as we know from the occurrence of anthracosis and other forms of pneumoconiosis. A similar resistance is offered by the tissues of the nose to the entrance of microorganisms. Considering the number of bacteria contained in the air, and the great quantity of air that passes through the nose, we would expect to find a very large number of microorganisms in the nasal chambers, as it is probable that they do not penetrate into the deeper air-passages. Opinions are divided on the fate of germs introduced into the nose, both as to the depth to which they penetrate into the nose and as to their behavior therein. While some investigators state that the nose is a playground for all kinds of bacteria, others, like Thomson and Hewlett,[1] have recently advanced the theory that the germs are arrested in the vestibule, and only in exceptional cases and in

[1] "Medico-Chirurgical Transactions," vol. LXXVIII, 1895.

small numbers penetrate into the deeper portions of the nose.

The question whether the nose contains germs under normal conditions has a practical bearing. There have been found the staphylococcus pyogenes aureus, the pneumococcus of Friedländer, the streptococcus, the diplococcus of Fränkel-Weichselbaum, diphtheria bacilli, and countless other bacteria of less importance, and Straus has shown that tubercle bacilli are frequently present in the noses of healthy individuals living among phthisical patients. But it is a matter of everyday observation that injuries or operative wounds in the nose usually heal without causing any, or at any rate very little, general disturbance, in spite of the apparent danger from infection which should result from the presence of such large numbers of bacteria. The explanation of this want of virulence on the part of the germs in the nasal cavities has been sought in a bactericidal quality of the mucous secretions. This was assumed by Wurtz and Lermoyez,[1] but Thomson and Hewlett found that the nasal mucus is not directly bactericidal, although it arrests the growth of germs to a certain extent. It is idle, in the absence of exact proofs, to discuss this question of the bactericidal powers of the secretion. The most that can be said is that it is not a favorable soil, and hinders the development of the microorganisms more or less. The conditions in this respect are evidently analogous to those found in the oral cavity, which contains even a greater abundance of bacteria. The mere presence of germs is not in itself injurious to the nose; other factors must be taken into account: the number and virulence of the pathogenic germs which gain entrance; the disposition of the individual; and the presence of other bacteria, which either assist or retard the growth of the pathogenic varieties.

2 and 3. Warming and Saturation of the Inspired Air.—It has been proved by the experiments of Aschenbrandt and of R. Kayser,[2] that the temperature of the air-current during its passage through the narrow chambers of the nose is raised to from 25° to 35° C., depending on the external temperature. It would, however, be wrong to suppose that this function of the nose is indispensable for breathing; Kayser has shown that the inspired air is

[1] "Ann. des mal. de l'oreille," 1893, p. 661.
[2] "Pflüger's Archiv," vol. XLI.

warmed only half a degree less in mouth-breathing than in nasal respiration, and that after tracheotomy the trachea and bronchi are quite capable of warming the air to 30° C.—the average temperature imparted to it in the nose —without injury to the lungs. Gaule suggests that the abundant supply of blood-vessels in the nose, and the property possessed by them of changing their volume, enable them to adapt themselves more easily to the thermic changes of the outside air.

A far more important function of the nose is to supply the necessary moisture to the inspired air—a function which the mouth is unable to perform. The nose thus relieves the bronchi and lungs of an onerous duty, which falls on them to a much greater degree if respiration is performed through the mouth.

To enable it to supply the required amount of moisture the nose is endowed with unusual secretory activity. The latter is derived in part from the abundant supply of serous and mucous glands and from an extensive system of lymphatics; also in part from an "irrigation-system, which keeps the epithelium constantly supplied with the necessary amount of moisture." The source of this special system is to be sought, according to Schieffendecker, not in the lymphatic vessels, but in the lymph-spaces of the tissues, the moisture making its way to the surface through the basal canaliculi, which pierce the basal membrane and emerge between the epithelial cells.

4. In addition to these functions of the nose, there are other **protective contrivances** of less importance **in the upper air-passages,** which prevent the entrance of deleterious substances into the lungs. Thus, the sense of smell serves to protect the organism by testing the inspired air and guarding the lungs against the entrance of substances which can be recognized by their odor. This protection is, after all, a faulty one, as there are many odorless gases which are injurious to the lungs and can not be detected in the inspired air by the sense of smell.

In the physiologic reflexes the body possesses another means of ridding itself of coarse particles of matter that have gained entrance to the nose with the inspiratory air-current, the mucous membrane bringing the sneezing reflex into action.

It is thus seen that under normal conditions respiration is effected through the nose, the lips being closed and the oral cavity occluded anteriorly and posteriorly by means of the tongue. The latter completely fills the oral cavity during nasal respiration, its tip being pressed against the upper teeth and the dorsum and edges fitting against the palate and alveolar processes, while the base of the tongue arches upward and is closely applied to the soft palate, so that the oral cavity is hermetically closed and shut off from the pharynx.

The question presents itself, whether the mouth is capable of supplying the functions of the nose in preparing the air for respiration, or whether mouth-breathing is injurious to the organism; and the answer must be that the oral cavity is not in any way adapted to replace the nose in the act of breathing. The width of the oral cavity is such that the air-current encounters no resistance, and consequently its progress is not retarded, as it is in the narrow passages of the nasal cavity, and no time is afforded for purification and saturation. The less abundant vascular supply and the absence of cavernous tissue (the amount of blood in which is regulated by the external temperature, and thus tends to maintain the required degree of temperature in the nose); the absence of an abundant watery secretion in the oral cavity; the nature of the epithelium in the mouth, which is of the squamous variety, and therefore incapable, in contradistinction to the ciliated epithelium in the nose, of removing automatically any deleterious substances in the air-current all these structural differences combine to make the mouth unfit to supply an air-current which would be other than injurious to the organism.

II. Pharynx and Larynx as Respiratory Pathways.—When the air reaches the pharynx and larynx, after passing through the nose, it has undergone the necessary preparatory changes for its entrance into the lungs, and needs no further alteration of any moment. If any particles of dust enter the larynx with the inspired air, they are promptly expelled by the ciliated columnar epithelium. But the pharynx and larynx are nevertheless supplied with a protective apparatus capable of preventing the passage of foreign bodies in either direction—into the postnasal space and the nose, or into the trachea and deeper air-passages; and it is called into activity whenever food is taken, to

guard the air-passages against the invasion of food particles. The oral cavity is completely shut off from the rhinopharynx by the application of the soft palate against the posterior pharyngeal wall, but the larynx is not entirely occluded during deglutition, the bolus of food gliding easily into the esophagus over the arching dorsum of the tongue (which guards the entrance to the larynx), so that the action of the epiglottis in closing the larynx is not absolutely indispensable. If a foreign body, however, does get into the larynx, the glottis immediately closes,—as it does always at the slightest touch,—and the offending particle is expelled by coughing.

DISEASES OF THE LUNGS DUE TO DISTURBANCES OF THE PHYSIOLOGIC FUNCTION OF THE UPPER AIR-PASSAGES.

In returning from this physiologic digression to the discussion of the influence exerted on the respiratory organs by disease of the upper air-passages, I shall adopt a classification in which the first place is accorded to those diseases of the lung that develop in consequence of disturbances of the function of the upper air-passages.

Such disturbances may arise because the respiratory air-current can not make its way through the nose, so that mouth-breathing becomes necessary. The obstruction may be situated in the nose or in the postnasal space. Any one of the following conditions may be present, and necessitate mouth-breathing: Hyperplasias and tumors in the nose; structural anomalies in the framework of the nose obstructing the lumen, caused by deviation of the septum, by ridges on its surface, or by abnormal bulging or cystic formations in the muscles; occlusion of the posterior nares by tumors in the postnasal space, and especially by adenoid growths on the vault of the pharynx. The evil effects of mouth-breathing first manifest themselves in the mucous lining of the pharynx and larynx, which becomes dry because the air has not been properly prepared and saturated. Dust particles are deposited first on the mucous membrane of the mouth and oral pharynx—which is covered only with squamous epithelium—and later make their way into the larynx and deeper air-passages. The constant irritation of the dry and unpurified air coming in contact with the mucous membranes of the upper and lower

air-passages gives rise, as we can easily understand, to chronic catarrhal conditions. Thus it is found that mouth-breathers, as represented typically by children in the early stages of enlarged tonsils, are prone to become the subjects of catarrh of the upper air-passages, of recurring pharyngeal and laryngeal catarrh, and of acute bronchial catarrh; while if the condition continues, they usually develop chronic bronchitis, which can be permanently cured only by restoring nasal respiration.

In this way we can frequently explain the chronic catarrh which is seen almost constantly in children of scrofulous habit, in whom the hypertrophy of the lymphatic elements in the postnasal space is followed by occlusion of the posterior nares. Mouth-breathing is, however, not the only precursor of chronic catarrh in the deep air-passages; the condition frequently develops as a sequel to pathologic alterations in the nose itself, provided they are sufficient to render it unfit to afford the necessary protection to the lungs. In atrophic conditions of the nose, coupled, as they are, with metaplasia of the epithelium, foreign bodies contained in the inspired air cling to the walls of the cavities, and eventually penetrate into the deep air-passages. In examining persons afflicted in this way, whose work obliges them to breathe impure air, a mere inspection of the nose, pharynx, and larynx shows the dust-particles, whether mineral or vegetable, as, for instance, coal-dust and flour, clinging to the mucous surface, and it is easy to understand that these dust-particles may be carried down with the inspiratory blast and settle in the bronchi. Such morbid changes must necessarily favor the development of the various forms of pneumoconiosis, especially anthracosis and chalicosis.

Disturbances of the sensibility and of the reflex activity of the pharynx and larynx have an important bearing on the lungs and bronchi, as they facilitate the development of inspiration pneumonia. If there is anesthesia of the pharynx and larynx, and the cough reflex is diminished, it is easy for particles of food to enter the larynx; and when from anesthesia of the larynx the glottis fails to close, and there is no reflex cough, the offending body readily finds its way into the lower air-passages. Hence an inspiration pneumonia frequently complicates nervous affections, which, like diphtheria, are accompanied with disturbances of sensibility, or, like bulbar disease, with loss of reflexes.

On the other hand, it is worthy of remark that ulcerations and disturbances of mobility in the epiglottis do not, as a rule, interfere with deglutition, and therefore are not followed by inspiration pneumonia. Motor disturbances of the epiglottis are usually mechanical, being due to inflammation and swelling of the member, while ulcerations, which may be so great as to destroy the entire organ, usually result from syphilitic or tuberculous lesions. When either of these conditions is present, we should naturally expect that food particles would penetrate into the air-passages, the entrance to the larynx not being sufficiently occluded by the epiglottis. The fact that it does not happen is proof that the epiglottis is of no great importance as a protection to the larynx, its place being easily filled by the base of the tongue. If, however, the muscles of the tongue are paralyzed or atrophied, as in progressive bulbar paralysis, foreign bodies find no difficulty in entering the deeper air-passages.

DISEASES OF THE LUNGS IN MORBID CONDITIONS OF THE UPPER AIR-PASSAGES.

Diseases of the lungs may owe their origin to direct extension of disease of the upper air-passages to the trachea and bronchi. The causes are the same as those we have referred to in discussing the relations existing between diseases of the upper air-passages, chronic hypertrophic and chronic atrophic catarrh, and suppurative processes in the nose, in its tributary cavities, and in the postnasal space. Chronic bronchitis is the most frequent of the various sequels, and proves very obstinate, especially in cases of chronic suppuration in the tributary cavities of the nose, where the pus trickles down from the nasal pharynx into the deep air-passages and sets up a chronic irritation. The question of the relation between chronic catarrh of the upper and of the deeper air-passages has not received the attention it deserves; it is barely mentioned in the most general terms in connection with bronchitis, and the possibility of emphysema, bronchiectasis or fetid bronchitis being due to such causes is usually ignored. A paper by Sticker,[1] in which he establishes a causal relation between atrophy, or dry catarrh of the mucous membranes of nose

[1] "Arch. f. klin. Med.," vol. LVII.

and pharynx, and similar atrophic conditions in the trachea, bronchi, lungs, and pleura, is therefore worthy of notice. Genuine ozena, or rhinitis fœtida atrophica, is an atrophic process in the mucous membrane, shared to some extent by the skeleton of the nose, so that the turbinated bones are often entirely destroyed, and the nasal cavity attains enormous dimensions. The atrophy affects the glands and the erectile tissue, partly destroying both structures, but does not extend to the blood-vessels, which, on the contrary, according to recent investigators, become dilated. At the same time the ciliated cylindric epithelium is converted into horny squamous epithelium, giving the mucous membrane a dry, cicatricial appearance, which in the later stages also extends to the pharynx and larynx after the atrophic process has reached these parts. The disease is regularly accompanied by the secretion of a tenacious material, which dries, forms crusts, and gives off a characteristic penetrating fetor. The discharges make their way into the pharynx and larynx, and thence into the deeper air-passages, where they may set up chronic irritative conditions. Sticker has shown that, aside from the fact that diseases of the lungs may be caused by disease of the deeper air-passages secondary to a similar process in the nose and postnasal space, there is a general condition of which the atrophy of the mucous membrane is merely the superficial expression, and this he has called *xerosis of the mucous membranes*. This condition eventually leads to a wide-spread and more or less complete atrophy of all the mucous membranes in the body, and, as old age comes on, to a progressive increase in the size of the nasal and postnasal cavities, the larynx, the trachea, the bronchi, and, finally, the lungs. In cases of marked atrophy with ozena of the nose and pharynx experience teaches us to expect not only chronic bronchitis, but also emphysema and asthma-like attacks. If such a condition is met with in elderly persons who have all their lives suffered from chronic bronchitis due to ozena, it is readily explained as senile emphysema, or as a secondary emphysema, such as may develop gradually in chronic bronchitis. But how are we to explain such cases of pulmonary emphysema in young persons, barely twenty years old, with all the symptoms—especially dyspnea and cyanosis—which are found only in the severest grades of emphysema? I

remember particularly a healthy young farmer, twenty-one years old, who suffered from severe emphysema, and the only explanation that could be found was a marked ozena, which the patient said he had had for a long time. Great as was the temptation to establish a causal relation between the two diseases, there did not seem to be sufficient justification for doing so, if the lung disease was viewed merely as secondary to the disease in the postnasal space. Such cases confirm Sticker's theory of a general xerosis. The tendency of the finer bronchioles to a dry catarrh, leading to simple increased volume (volumen pulmonum acutum) and pulmonary emphysema, is interpreted by Sticker in his previously mentioned paper, as an expression of the general xerosis which primarily affects both the upper and lower air-passages.

But how does this xerosis originate? Is it a disease, brought on by external influences, by bacteria, by suppurations or excoriations in the nose, which is hidden under the disguise of what we call genuine ozena? Every one of the hypotheses that have been advanced to explain the occurrence of atrophic fetid rhinitis must be rejected as inadequate. All the various causes, from alterations in the epithelium, glands, and blood-vessels to the latest bacteriologic discoveries, to which the symptom-complex known as ozena has been attributed, while they may possess some accessory importance, are certainly not the primary etiologic factors. The production of a fetid secretion with a tendency to crust formation is not peculiar to ozena. Experience teaches that it may occur in any condition in which the capacity of the nose is increased, whether from destruction of the skeleton or from too severe treatment with the galvanocautery, or any form of atrophy of the mucous membrane. There is nothing new about Sticker's suggestion of syphilis as the cause of his mucous membrane xerosis. Störk repeatedly emphasized the probability of a causal relation between ozena and hereditary syphilis; but Sticker put the matter in a clearer light when he showed that the conspicuous local alterations in the nose are comparatively unimportant, and that the general xerosis is the primary condition, corresponding to a postsyphilitic destruction of the parenchyma, which may, by becoming complicated with chronic catarrh, give rise to ozena with its characteristic secretion.

It is at least worth while to examine this xerosis of the upper and lower air-passages, in order to determine whether chronic bronchitis and emphysema are really due to the same cause as the disease of the nose and postnasal space.

Among pathologic curiosities may be mentioned the cases in which corrosive fluids penetrate into the larynx, trachea, and bronchi. There have also been reported instances of fibrinous exudations due to vapor of ammonia being formed in the nose, pharynx, larynx, trachea, and even in the finest bronchioles (Hoffmann). Phthisical patients who had been treated for a long time with local applications of lactic acid on account of tuberculosis of the larynx have been known to expectorate ribbon-like shreds of mucus from the trachea and bronchi.[1]

Scleroma of the upper air-passages which, as Schrötter [2] and Baurowicz [3] had occasion to observe, extends to the bronchi and leads to stenosis, is a very rare condition, at least in this country. Schrötter's patient died of marasmus and fetid bronchitis ; Baurowicz's, of asphyxia brought on by stenosis of the bronchi.

ALTERATIONS IN THE UPPER AIR-PASSAGES IN DISEASES OF THE LUNGS.

The most important alterations in the upper air-passages occurring in the course of the various diseases of the lungs are those which are due to the irritation of the mucous membranes by the passage of the secretions. Any chronic disease of the lungs in which sputum is secreted is followed sooner or later by chronic laryngeal and pharyngeal catarrh, the intensity of which is in direct proportion to the amount and consistency of the expectorated material and to the amount of effort required to effect its expulsion. Hence, asthmatic and emphysematous patients, whose bronchi are filled with tenacious sputum which requires severe coughing and straining to remove, suffer more from inflammatory conditions of the upper air-passages than do those who have a simple bronchitis with watery secretions, which they can expel without straining the muscles of the neck and throat.

[1] F. A. Hoffmann, "Die Krankh. der Bronchien," in Nothnagel's "Spec. Path. u. Therap.," p. 135.

[2] "Mon. f. Ohr.," 1895, p. 149 *et seq.*

[3] "Arch. f. Laryng.," IV, p. 99.

A form of ascending catarrh of the air-passages has been described, beginning with bronchitis and terminating in acute laryngitis and pharyngitis. Emphysematous individuals suffer from congestive catarrh, and are prone to have hemorrhages.

According to Heinze [1] and Landgraf,[2] croupous pneumonia is sometimes followed by laryngeal complications. Among fifty cases of laryngeal ulceration, Heinze found one in which the vocal cords were ulcerated in the course of croupous pneumonia, and he states that the ulcerations were not tuberculous. Landgraf analyzed eighty cases of croupous pneumonia, and found two cases of ulceration in the larynx. In both cases the primary disease was severe,—one being a bilious form and the other being accompanied by severe sensory phenomena,—and he attributes the ulcers to the dyspnea, "interpreting them as decubital ulcers analogous to typhoid ulcers": "The closure of the glottis which precedes the cough—in other words, the pressure on the vocal processes and free borders of the vocal cords—led to the formation of ulcers in these situations."

The most frequent complications of lung diseases in the larynx consist in paralysis, the occurrence of which after disease of the lungs and pleura is explained by the course of the recurrent laryngeal nerve. The plications [3] which form in the pleura over the apices in chronic inflammations are very apt to include the nerve, especially on the right side, where such a complication is favored by the relation of the nerve to the subclavian artery; and, on the other hand, indurations of the apex may during cicatrization exert traction on the nerve. Paralyses of the right or left recurrent are most frequent in chronic indurative pleuritis, and produce permanent alterations in an acute left pleuritis. Schrötter [4] once observed a paralysis, which disappeared after ten days, and infers that it was a case of inflammatory edematous infiltration of the pleura. Paralysis rarely develops in pleural exudations. Moeser [5] claims to have observed that patients with pleural exudations, particularly when there is a copious effusion of fluid, and oftener

[1] "Die Kehlkopfschwindsucht," p. 87.
[2] "Charité Ann.," XII, p. 244 *et seq.*
[3] Comp. Gerhardt, "Virch. Arch.," XXVII, p. 76.
[4] "Die Krankheiten des Kehlkopfes," p. 414.
[5] "Arch. f. klin. Med.," XXXVII, p. 570 *et seq.*

on the right than on the left side, present peculiar motor disturbances of the vocal cord on the corresponding side, which almost always consisted in "diminished power of abduction"; but his observations are not sufficiently convincing, and do not present the typical picture of a pronounced paralysis of the recurrent. Nor is there more plausibility in the attempts to explain a paralysis occurring in a left pleural effusion by the downward displacement of the heart exerting direct traction on the aorta and the recurrent nerve; or one occurring in a right effusion by the displacement of the heart to the left and the consequent traction on the vessels of the right side, especially the subclavian artery and the recurrent nerve which winds around it.

Chronic conditions in the apex of the right lung—such as tuberculous consolidation or chronic induration from the inhalation of dust—may produce recurrent paralysis. Compared to the great frequency of pulmonary tuberculosis, paralysis due to this condition is exceedingly rare,—Jurasy [1] saw only three cases, which he did not describe in detail,—and the diagnosis can not even be established with absolute certainty during life, for, as will be described later, the nerve may be pressed upon by swollen lymph-glands within the thorax. The same is true of recurrent paralysis in anthracosis, examples of which are found in cases of Bäumler [2] and Kohn,[3] in which the cause of the paralysis turned out to be an adhesion of the recurrent nerve to an indurated, deeply pigmented and contracted bronchial gland.

ALTERATIONS IN THE UPPER AIR-PASSAGES IN DISEASES OF THE MEDIASTINUM.

Before closing the section on the respiratory organs, mention should be made of alterations due to disease in the mediastinum and changes in the thyroid gland.

The inferior laryngeal nerves traverse the mediastinum, and are, therefore, exposed to injury from disease in that locality.

In the case of mediastinal tumors the left nerve, on ac-

[1] "Krankh. der ob. Luftwege," p. 476.
[2] Comp. Bäumler, "Arch. f. klin. Med.," XXXVII, p. 231 *et seq.*
[3] "Münch. med. Wochen.," 1895, p. 1131.

count of its position in the lowest part of the mediastinum, is most apt to be included within the growth. But it also happens, as in Michael's case,[1] that in spite of its more superficial course the right recurrent nerve becomes embedded in the tumor.

Paralysis of the inferior laryngeal nerves may follow disease of the bronchial lymph-glands, and of the glands belonging to the group of *ganglions trachéo-latéraux* (Barety),[2] designated as *ganglions péritrachéo-laryngiens* by Gougenheim and Leval Picquechef,[3] which occupy the groove between the trachea and the esophagus, and therefore come into close relation with the recurrent nerve, which emerges from the mediastinum in the same situation on each side of the body.[4]

These glands often become enlarged in scrofulous children after bronchopneumonia and other forms of chronic pulmonary catarrh; they are also found to be enlarged in tuberculosis and in melanotic degeneration. An example of the latter has recently been mentioned in Bäumler's case, while the literature of tuberculosis of the bronchial glands has lately been worked up by Fronz.[5] If suppuration take place in the glands, the abscess sometimes ruptures into the trachea, and the pus is evacuated into the larynx, as observed by Massei [6] in several cases.

Besides pressing on the nerve-trunk, mediastinal tumors may push the larynx to one side or the other and compress the trachea. Such a stenosis from compression of the walls is sometimes seen in the trachea with the laryngoscope; rotation of the trachea and larynx may take place, so that the glottis appears oblique in the laryngoscopic image. Struma is a more frequent cause of tracheal stenosis than mediastinal tumor, a large percentage of all stenoses being due to goiter. Rosenberg [7] found that out of fifty-four cases of tracheal stenosis, thirty-eight were caused by struma.

The occurrence of vocal cord paralysis in struma de-

[1] "Die med. Wochen.," 1895, No. 25.

[2] Comp. F. A. Hoffmann, "Erkrankungen des Mediastinum," 1896, p. 30.

[3] "Ann. des mal. de l'oreille," etc., 1884.

[4] An excellent illustration of these anatomic relations is found in Gougenheim—Glover's "Atlas of Laryngology," I, XIX.

[5] "Jahrb. f. Kinderheilk.," vol. XLIV.

[6] "Rev. de lar., d'ot et de rhin.," 1897, No. 7.

[7] Heymann, "Handb. der Laryng.," I, p. 568.

pends more on the position than on the bulk of the enlargement; if the lateral lobes are affected, pressure is exerted on the nerves, and paralysis frequently results.

Among the structures in the mediastinum which may affect respiration unfavorably the thymus gland must also be mentioned. In recent times, cases of sudden death in children have been attributed to hyperplasia of the thymus,[1] just as the inspiratory stridor of the new-born is now generally acknowledged to be referable to the same cause. Siegel[2] and Avellis[3] have contributed valuable descriptions of the clinical picture presented, supplemented with full histories of the cases.

A signal proof of the relation between inspiratory stridor and enlarged thymus is afforded by Rehn's case, reported by Siegel, and by a case operated on by König, in which recovery was brought about by exposing the gland, after resection of the sternum, and fixing it to the cervical fascia in one case, and by extirpating a part of the gland in the other. In the typical case there is labored, groaning respiration, not occurring in paroxysms but persistent; the dyspnea sometimes amounts to violent choking fits with cyanosis, so that the affection (which is also called asthma thymicum) has often been erroneously described as laryngismus stridulus. Since the clinical feature is not a spasm of the vocal cords, but a compression of the trachea and bronchi, the term inspiratory stridor is the most scientific, and should be applied at least to cases in which the diagnosis of a thymic origin for the dyspnea is not quite clear. This compression, as M. Schmidt[4] observed in a woman, twenty-five years old, who had suffered from inspiratory stridor during infancy, may be permanent.

3. RELATIONS BETWEEN THE UPPER AIR-PASSAGES AND THE EARS.

The interdependence of the upper air-passages and the ears depends, in the majority of cases, on the communication established by the Eustachian tubes, which open into

[1] Avellis, "Arch. f. Laryng.," VIII, p. 159.
[2] "Berlin klin. Wochen.," 1896, No. 40.
[3] "Münch. med. Wochen.," 1898, Nos. 30 and 31.
[4] Cited by Avellis.

the lateral walls of the pharynx, and their function in connection with the middle ear. Hence, to understand the mechanism of secondary ear affections, when the primary disease focus is in the upper air-passages, the following points, which will later be described more in detail, must be borne in mind.

The Eustachian tube ventilates the middle ear, and regulates the tension by equalizing the differences that may arise between the atmospheric pressure on the tympanic membrane in the external meatus and in the postnasal space, and the pressure of the air imprisoned in the middle ear. Whenever the equilibrium is disturbed, auditory disturbances result, which have their seat in the tympanic membrane and in the sound-conducting apparatus.

Such disturbances may follow disease in the upper air-passages, obstructing or occluding the pharyngeal orifice, or interfering with the action of the palatal muscles which effect the opening and closing of the tube.

The Eustachian tube represents the path by which disease of the pharyngeal vault spreads by continuity to the middle ear; the mucous membrane of the tube becomes involved in any disease affecting the mucous membrane of the nose and pharynx; or, in other words, the tube represents the channel through which infection reaches the middle ear from the upper air-passages.

THE EFFECT OF DISTURBANCES OF THE NORMAL FUNCTION OF THE EUSTACHIAN TUBE.

For the proper comprehension of this relation a few introductory remarks are required to explain the mechanism by which the normal tube neutralizes the variations in pressure of the atmospheric air in the middle ear. The tube does not keep up a constant communication between the two air-chambers—that of the pharynx and that of the middle ear. In the state of rest its pharyngeal extremity remains closed,[1] and is opened only when the muscles of the palate and pharynx, which are devoted to its service, are brought into action. As the opening of the tube is effected by the tensor veli and levator veli muscles, the pharyngeal orifice must be opened whenever these muscles contract, which happens

[1] Hammerschlag, in "Wien. med. Wochen.," 1896, Nos. 39 and 40, makes the assertion that the tube is normally open.

regularly and frequently, accompanying the act of deglutition. The opening of the tube is, therefore, under the control of the will power, and we can equalize any disturbances of the pressure equilibrium by the simple act of swallowing, which establishes a communication between the postnasal space and the middle ear, through the opened tube. We instinctively take advantage of this phenomenon whenever a change in the atmospheric pressure takes place, as in climbing high mountains, during explosions and loud detonations, and, artificially, for therapeutic purposes, by means of pneumatic chambers.

What, then, is the result if the tube fails to maintain the equilibrium? What happens when the pressure is greater in the pharynx than in the middle ear, either because the pressure of the outside air has been raised or because the pressure of the air in the middle ear has been lowered?

Valsalva's experiment, which consists in artificially raising the air pressure in the postnasal space by making a forced expiration with the nose held shut,—when the pharyngeal orifice of the tube is forcibly opened and the air escapes into the middle ear,—would appear to indicate that the orifice opens automatically whenever the pressure in the pharynx is even slightly increased. This is not the case, however. On the contrary, a rise in the atmospheric pressure has the effect of closing the tube tighter than ever, for we know from practical as well as experimental observation that increased pressure in the pharynx brings the membranous wall of the tube into closer apposition with the cartilage, thus forming a kind of valve, which shuts the canal off from the middle ear. The closure effected in this way may be so obstinate that an ordinary act of swallowing is unable to overcome it.

A rise of pressure takes place regularly in the pharynx, independently of changes in the atmospheric pressure, whenever the tube remains closed for any length of time, and the air imprisoned in the cavities of the tympanum undergoes rarefaction.

It follows, therefore, that a fall in the tympanic pressure occurs in all diseases in which the pharyngeal orifice of the Eustachian tube is occluded. In explanation of this phenomenon we can not do better than quote the words of Bezold:[1] "In the middle ear, as in all vascular, air-con-

[1] "Berl. klin. Wochen.," 1883, No. 36.

taining cavities, the volume of air diminishes whenever free communication with the atmosphere is interrupted, because the oxygen enters into chemic combination with the blood and the amount of CO_2 given up is not enough to compensate for the loss in volume."

These pressure variations give rise to a series of clinical pictures which are included under the general term of acute or chronic middle-ear catarrh, and have as their most prominent symptom retraction of the tympanic membrane —a purely mechanical result of the increased pressure in the external auditory meatus. In the otoscopic image this abnormal position and the curvature of the tympanic membrane find their expression in the absence of the cone of light from its normal situation; in the presence of irregular reflexes; in displacement of the handle of the malleolus, which assumes a more horizontal position, or eventually even disappears behind the posterior fold; and, finally, in a marked projection of the short process and handle of the malleolus from the retracted membrane. The subjective symptoms are diminished auditory acuity and tinnitus aurium; occasionally the patient complains of pain. The question whether the occasional occurrence of exudation in conditions of diminished tympanic pressure is due solely to *hyperæmia ex vacuo* can not be answered in the affirmative in every case. Although the possibility of such an origin deserves consideration, the fact that *hyperæmia ex vacuo* is not by any means a regular accompaniment of occlusion of the tube is sufficient proof that other factors must also be operative to produce an effusion, and it is safe to assume the occurrence of an irritative inflammation of the mucous membrane. The idea of *hydrops ex vacuo* is therefore limited in its application, and must in many cases give way to the theory of an inflammatory exudate. This view is confirmed by clinical experience, since it is found that chronic occlusion of the tube, which produces the greatest diminution of density, is not, as a rule, followed by exudation. On the other hand, in almost all cases of occlusion occurring after acute inflammations an exudate is formed which betrays its inflammatory nature in the otoscopic image by a simultaneous swelling and congestion of the deeper layers of the membrana tympani, and can be ascribed only to inflammatory swelling of the mucous membrane of the middle ear, with secondary extension to the mucosa of

the membrana tympani. It follows, therefore, that the exudate is as much a symptom of middle-ear inflammation, or the time-honored "otitis media catarrhalis," as of occlusion of the Eustachian tube. The results of bacteriologic investigations of this condition are not uniform enough to throw much light on the subject. Kanthack [1] found a great variety of pathogenic organisms in exudations following occlusion of the tube, while Scheibe [2] and Brieger [3] deny the presence of these organisms, and consider the "exudate ex vacuo" sterile; hence we are not as yet justified either in adducing the finding of microorganisms as proof of the inflammatory character of the exudate, or in denying it on the strength of a negative bacteriologic result.

There is also a possibility of the pressure equilibrium being disturbed by an *excess of pressure in the middle ear*, most frequently due to a fall in the atmospheric pressure. Its pathologic significance is slight compared to the opposite condition. The rise in pressure is readily equalized by the tube, because, as previously described, there is no occlusion of the pharyngeal orifice by the air pressure, and the equilibrium is therefore easily restored by the act of deglutition. Hence a gradual fall in pressure, such as is experienced in balloon ascensions and in mountain climbing, is easily borne, because the excess of pressure which at first occurs in the middle ear soon accommodates itself to the surrounding conditions. On the other hand, when the external pressure is suddenly removed, the excessive pressure in the middle ear is very apt to produce disturbances in the auditory organs by rupture of the membrana tympani. This is apt to occur when men are released from a caisson without the necessary precautions. Sudden rise of pressure in the middle ear is sometimes produced by blowing the nose, if the pharyngeal orifice is forcibly opened by a sudden increase in pressure in the postnasal space, allowing the air to escape through the tube. It is particularly in cases of nasal obstruction, when the expiratory blast can not escape and becomes imprisoned in the postnasal space, that violent blowing of the nose is apt to be followed by serious consequences in the ear, by producing hemorrhages or rupture of the membrane, especially if the latter is diseased or atrophic.

[1] "Zeitschr. f. Ohr.," XXI. [2] "Zeitschr. f. Ohr.," XXIII.
[3] "Beitr. z. Ohrenheilk.," p. 59.

It is still an open question whether the tinnitus aurium which occasionally occurs in gradual changes of the external air pressure originates within the organ of hearing, or whether it is due to other causes. The subjective noises heard during balloon ascensions and mountain climbing are naturally attributed to the variations in pressure between the middle ear and the external air. But if we study the clinical picture of so-called mountain sickness, we are struck with the predominance of the circulatory phenomena, the markedly accelerated pulse, the dyspnea, and local symptoms of flashes and subjective noises in the ears; we are irresistibly led to attribute the ocular and aural phenomena to the vascular disturbances, and not to alterations of the special sense organs.

It may be well in this connection to call attention to the fact that similar phenomena, though somewhat milder in character, occur in the treatment of heart and lung diseases with rarefied and compressed air, when the patients are entering or leaving the pneumatic chamber. A certain degree of caution is therefore advisable in the case of individuals whose hearing is not quite perfect, especially such as are troubled with tinnitus aurium.

Schwartze's [1] observation that "many persons with incurable middle-ear sclerosis experience relief during a protracted stay in high Alpine health resorts, on account of their freedom from the distressing tinnitus aurium, and the marked improvement in the hearing" has not been explained. But is it not permissible to assume that the aural symptoms are due to circulatory disturbances? For if the tinnitus is really a vascular murmur within the ear, would it not be relieved by the beneficial effect of the altitude on the heart?

DISTURBANCES OF THE FUNCTION OF THE EUSTACHIAN TUBE DUE TO ALTERATIONS IN THE UPPER AIR-PASSAGES.

The diseases of the upper air-passages that diminish the permeability of the tubes are principally those which are accompanied by swelling of the mucous membrane. The relation existing between the nose and the postnasal space is a very intimate one, and very few diseases have their

[1] "Die chirurg. Erkr. des Ohres," p. 169.

origin and exclusive seat in the postnasal space without involving the nose. The great majority of pharyngeal diseases arise, as we have already stated, by extension from the nose, so that the importance of rhinology in the study of diseases of the ear is easily explained.

Any *acute catarrh* in the upper air-passages may lead to intumescence and occlusion of the pharyngeal orifice, and thereby produce a fall in the pressure of the middle ear. As soon as the swelling subsides and the tube again becomes patulous, the morbid symptoms usually disappear without treatment. In *chronic catarrh*, on the other hand, as the hypertrophy of the mucous membrane does not, like the hyperemia in acute catarrh, tend to disappear spontaneously, the changes produced in the middle ear by occlusion of the tube are of a more lasting character. The continued tension of the membrana tympani leads to atrophy, and the persistent retraction disturbs the normal relation of the ossicles, which then exert a constant pressure on the fenestra ovalis.

It is fair to assume that, in consequence, the muscles of the ossicles—the tensor tympani and the stapedius—are thrown into a state of permanent contraction, and probably atrophy from disuse ; while, as a result of the *hyperæmia ex vacuo*, a chronic inflammation of the mucous membrane of the tympanum develops, giving rise to morbid conditions which can not be distinguished clinically from catarrh of the middle ear due to other causes. These conditions are in great need of anatomic and clinical investigation ; in fact, the concept of middle-ear disturbance by occlusion or obstruction of the tube has never been clearly differentiated from the idea of inflammatory middle-ear catarrh, and the various views advanced in the text-books descriptive of middle-ear catarrh of inflammatory and noninflammatory origin merely add to the confusion.

Any and all diseases of the nose and postnasal space which are followed by obstruction of the nasal passages lead to passive hyperemia in the mucous membranes, which in turn produces occlusion of the Eustachian canal. The recognition of this important fact is comparatively recent, and since the causal relation between these disturbances and the interference with nasal respiration by the presence of adenoid growths was definitely established the attention of clinicians has been directed to the significance of nasal

stenoses in occlusions of the Eustachian tube. The interference with nasal breathing may be due to a number of conditions within the nose, as hypertrophy of the mucous membrane, mucous polypi, tumors, syphilitic or tuberculous infiltrations, foreign bodies, etc. There may be a congenital narrowing of the nasal cavity from deformity, and hyperplasia of the septum or abnormal curvature of the turbinated bones. The obstruction may be situated in the postnasal space, and may take the form of hypertrophied pharyngeal tonsils, tumors, syphilis, or tuberculosis occluding the posterior nares. Hence the recognition and removal of any obstacle to nasal respiration should constitute an integral part of every examination and treatment of the ears. There can be no hope of curing the ear affection before the causes which are responsible for the congestion of the mucous membrane have been removed and the permeability of the tube has been restored.

If I have included *hypertrophy of the pharyngeal tonsils* or *adenoid vegetations* among the diseases which produce hyperemia and swelling of the mucous membrane, with occlusion of the tube by interfering with nasal respiration, it is because I believe the occlusion is due to a general "adenoid habit" of the nose and pharynx, rather than to the direct mechanical intrusion of the pharyngeal tonsil. The "adenoid habit" manifests itself in the rhinoscopic image as a hyperplasia of the entire lymphatic ring of Waldeyer ; the follicles in the posterior pharyngeal wall and in the longitudinal folds on each side of the pharynx are more numerous, and are intensely red and swollen. Hyperplasia of Rosenmüller's crypts and of the anterior fold of the tube may develop as the manifestation of a general hyperplasia of all the lymphatic elements entering into the formation of the so-called pharyngeal lymphatic ring ; hyperplasia of these structures necessarily favors the occlusion of the tube by compressing the orifice.

Two forms of adenoid enlargement are distinguished : a diffuse, cushion-like hyperplasia, and a villous variety consisting of finger-like projections or true vegetations. Their usual seat is the roof and upper portion of the posterior wall of the pharynx, so that they fill the upper part of the postnasal space more or less completely, and whenever they hang down below the level of the upper margin of the posterior nares, the latter are obstructed and nasal breath-

ing is interfered with. As the vegetations usually spring from the median line, they are not, when at rest, in contact with the lateral walls of the pharynx and therefore do not occlude the orifices, as we are frequently able to demonstrate in the postrhinoscopic image; but whenever the palatal and pharyngeal muscles contract, as in swallowing, retching, and similar movements, the lumen of the postnasal space is constricted and the enlarged growths are forced against the lateral walls of the pharynx, thus giving rise to periodic occlusion of the tube. The adenoid tissue is not the soft, gelatinous mass that it is sometimes compared to, but is comparatively firm, and returns to its normal position of rest, dependent on gravity, as soon as the constrictors of the pharynx and the tensores and levatores palati relax and the postnasal cavity regains its normal volume. But it is not clear to me how a momentary occlusion of the orifice can have the same effect as a permanent one, and I therefore consider the hyperemia of the entire mucous membrane the most important factor in the production of aural complications.

Paralysis of the muscles of the soft palate, especially of the levator veli palatini and tensor veli palatini,—muscles which effect the opening of the Eustachian tube,—is followed by permanent occlusion, with the usual appearances of the membrana tympani. The action of the muscles may be similarly affected by tumors, by syphilitic, tubercular, or other kinds of ulcerations or their scars, and by cleft palate, so that these conditions are also occasionally accompanied by middle-ear disease.

DISEASES OF THE MIDDLE EAR DUE TO INFECTION FROM THE POSTNASAL SPACE.

The cartilaginous portion of the Eustachian tube is lined with ciliated columnar epithelium, the ciliary current being directed toward the pharynx, which is replaced at the isthmus by cells of the same type as that of the middle ear. Since, therefore, the mucous membrane of the tube is a direct continuation of the epithelium of the postnasal space, we can readily understand that an inflammatory process beginning in the latter is not arrested at the pharyngeal orifice, but is continued into the tube itself, and may be followed by acute inflammation of the mucous membrane

of the middle ear. Next to acute rhinitis and pharyngitis, the most important inflammations in the etiology of otitis media are those which occur in the *acute exanthemata.* These will be discussed elsewhere.

In addition to the ordinary inflammations of the mucous membrane of the tube and middle ear, we observe *acute suppurative otitis media* in the train of acute nasal and pharyngeal diseases. As we may have either a simple or a suppurative inflammation without any apparent external reason, we are forced to assume a different behavior of the mucous membrane of the middle ear in regard to the invading pathogenic germs to explain the occurrence of suppuration in the middle ear through the channel of the Eustachian tube.

It is well known, as has been mentioned, that the nose and postnasal space harbor a multitude of microorganisms. Their presence in the healthy organism appears to have no significance, perhaps because of a bactericidal property of the mucous secretion which destroys the virulence of the pathogenic germs and prevents their further development. It may also be assumed—and has, in fact, been practically demonstrated experimentally by Zaufal, Kanthack, Scheibe, and others, in spite of the differences in the individual results—that the middle ear normally contains bacteria which may, under favoring circumstances, regain their virulence.

The number of bacteria in the middle ear and the liability of the organ to infection depend on the condition of the epithelium lining the tube and the size of the lumen. If the ciliated epithelium is intact, it enables the tube to rid itself of any deleterious substances, and thus forms a protection against invasions from the pharynx. Since any inflammatory alterations, be they acute or chronic, which destroy the integrity of the epithelium tend to remove this natural protection, they will naturally be accompanied by inflammation and suppuration in the middle ear.

The question whether abnormal dilatations of the tube may produce pathologic conditions in the ear by affording an easier entrance to pathogenic germs deserves passing mention.

In catheterization of the tube the nature of the blowing noise, and the strength of the concussion-note afford a clue to the size of the lumen. But, in addition to this, other

signs have been noted, depending on the respiration, which point to abnormal dilatation and permanent patulousness of the tube.

Respiratory movements have been observed *in the tympanic membrane* by Lucae,[1] Schwartze,[2] Wagenhäuser,[3] and Ostmann;[4] the membrane retracts during inspiration and bulges toward the external meatus in expiration. These observations were, however, always made on atrophic or cicatricially contracted membranes, which respond to a much slighter pressure than would a tense healthy membrane. According to Ostmann, it is sometimes possible, with the aid of a tube inserted into the external meatus, to hear an *inspiratory* and *expiratory murmur*, even in healthy individuals during quiet nasal respiration. Finally, Lucae's manometric experiments called attention to the occurrence of *pressure variations in the external auditory meatus synchronous with the respiratory movements*. Ostmann, it is true, obtained varying results when he tried the same experiments; although he observed a constant variation of about ½ of a mm., synchronous with the pulse-beat, he could not demonstrate a constant coincidence with the respiratory movements.

The first-mentioned phenomenon—respiratory movements of the tympanic membrane—is undoubtedly to be attributed to abnormal dilatation and permanent patulousness of the tube, while the second is of no value in the diagnosis of these conditions.

Abnormal dilatation of the tube is practically a constant feature of atrophic catarrh of the nose and pharynx, rhinitis fœtida atrophica, and these conditions are regularly accompanied by disease of the middle ear, either in the form of sclerosis of the middle ear or chronic suppurative otitis media.

Sclerosis occurring in atrophic rhinitis is caused by a disease of the mucous membrane analogous to the dry catarrh of the upper air-passages. The histologic changes in otitis media sclerotica closely resemble those of "xerosis" of the mucous membranes of the upper air-passages (Sticker), so that the middle-ear affection must be interpreted as a process analogous to atrophic rhinitis and pharyngitis. This was pointed out several years ago by Abel, when he

[1] "Arch. f. Ohr.," vol. II.
[2] "Arch. f. Ohr.," vol. II
[3] "Arch. f. Ohr.," vol. XXI.
[4] "Arch. f. Ohr.," vol. XXXIV.

demonstrated his bacillus mucosus ozœnæ in the middle ear, although that discovery seems to me to be of little importance, in view of the questionable connection between this bacillus and the development of ozena.

It may be said that suppuration in the middle ear is principally due to the greater ease with which pathogenic germs can gain entrance when the tube is dilated, and to metaplasia of the epithelium.

Ostmann [1] has called attention to another pathologic change at the pharyngeal orifice which may produce dilatation of the tube. The lateral wall of the tube is provided with a pad of fat, which normally acts as a natural protection by facilitating the close application of the lateral to the median wall and thereby closing the tube. In emaciated individuals this pad is so much reduced that the tube is not perfectly closed, and there is a greater tendency to infection of the middle ear from the pharynx. Ostmann believes this to be the explanation of tubercular suppuration in the middle ear, which develops in phthisical patients when the general condition is weakened, and in the fourth or fifth week of typhoid, when the nutrition of the patient is much reduced.

From a practical standpoint the infection of the middle ear through the introduction of infectious material into the tubes by therapeutic measures is extremely important. It may occur—by the current of air carrying mucus and pus from the nose, postnasal space, or orifice of the tube into the ear—during the performance of Valsalva's experiment or violent blowing of the nose with the nostrils closed, which has the same effect, and in Politzer's method of inflating the tympanum, which consists in blowing air through one side of the nose while the nasopharynx is shut off from the oral cavity and the anterior nares are closed, so as to raise the air pressure in the postnasal space and force open the pharyngeal orifice of the Eustachian tube. Another danger of infection of the middle ear from the pharynx arises from the use of nasal douches in hypertrophic conditions of the nasal mucous membrane. As the water enters the nasal cavities under considerable pressure, and can not escape through the nose on account of the swelling of the membrane, it is dammed up in the postnasal

[1] "Arch. f. Ohr.," XXXIV, p. 188, etc.

space, and being under pressure, easily makes its way into the middle ear. This is, therefore, an example of the improper use of the nasal douche, being in violation of the principle that the use of a nasal douche with low pressure is permissible only when both sides of the nose are sufficiently open to allow free access to and egress from the nose. Even when this rule is carefully observed there is a possibility of water reaching the middle ear, if during its passage the patient swallows or chokes, or performs a similar act which opens the Eustachian tubes and facilitates the entrance of the fluid into the middle ear.[1]

But as, in spite of the frequency of these harmful conditions and the presence of infectious mucus in the upper air-passages, infection of the middle ear takes place only occasionally and in certain cases, it is evident that the development of pathogenic germs is determined more by a favorable condition in the ear itself than by the fact of their gaining entrance through the tube. Such a condition is produced chiefly by acute inflammations of the mucous membrane, and we expect to find suppuration of the middle ear in acute coryza, in the acute exanthemata which are accompanied by rhinopharyngitis, and in the acute inflammation which follows the use of the galvanocautery in the nose; and we can not emphasize too strongly that air douches, as well as the ordinary nasal douche, are to be avoided in acute disease of the nose and throat with inflammatory changes in the Eustachian tubes.

4. THE EFFECT OF VARIOUS DISEASES OF THE RESPIRATORY ORGANS ON THE EARS.

Pain in the ears or pain radiating from the neck to the ears is a symptom occurring in a great variety of diseases of the upper air-passages. It occurs with the greatest regularity in all inflammatory diseases of the epiglottis and upper margin of the larynx which are accompanied by swelling, and is met with also in malignant tumors of the

[1] The rule which obtains in our country—viz., to introduce the tip of the nasal douche only into the side of the nose most obstructed, to allow free exit of the fluid from the more open side—would seem an important suggestion.—Ed.

larynx and upper portion of the esophagus. The physician sees many cases of tuberculosis with infiltration and ulceration of the mucous membrane covering the cartilages, and perichondritis of the epiglottis and arytenoid and cricoid cartilages, in which the patients complain of violent pain radiating to or localized in the ear. The pain is increased by any pressure or movement in the affected region, and usually attains its maximum intensity during the act of swallowing, making it very difficult to feed the patient properly. The greatest distress is usually complained of when the patient involuntarily goes through the act of swallowing, just as in any form of angina, and especially in tonsillar abscess, while the swallowing of slimy, semisolid food is a little less painful.

In carcinoma of the larynx pain radiating to the ears is practically a constant symptom ; it is usually of a paroxysmal character, like the lancinating pain of neuralgia. In the early stages of the growth the pain is dull and localized in the larynx, but radiates to the ears when the ulcerative stage is reached.[1]

Since the sensory nerves at the entrance to the larynx and in the deeper portions of the pharynx are branches of the vagus, and the external auditory meatus receives sensory fibers from the same trunk through the auricular nerve, the vagus must be the channel by which these reflex pains are transmitted. The reflex arc is very well developed, as is shown by the fact that irritation of the sensory filaments of the auricular nerve of the vagus in the external meatus—as, for instance, when a speculum is introduced—often brings on a fit of coughing.

Our information in regard to the relation existing between croupous pneumonia and purulent otitis media is not very definite and lacks clinical confirmation ; it amounts to this : suppuration in the middle ear is rare after croupous pneumonia, presents no distinct type, and its course is not different from that of any other form of purulent otitis media. In severe cases of pneumonia with high fever the tympanic membrane is found to be injected without exudation taking place, just as in other infectious fevers, especially typhoid. Again, as in other diseases characterized by great

[1] Comp. Fauvel, " Traité pratique des malad. du larynx," 1876, p. 707.

elevation of temperature, a chronic suppuration may temporarily subside during the fever, and the perforated membrane and mucosa of the tympanum appear dry and dark red in color; but there is no reason to suppose that these phenomena have any specific relation with the pneumonia.

Acute suppuration in the middle ear is occasionally produced by the diplococcus pneumoniæ of Fränkel-Weichselbaum. Netter,[1] as early as 1890, called attention to the frequent occurrence of the pneumococci in the pus found in the ears of little children at autopsies, and his findings have been confirmed by Rasch,[2] who found the pneumococci of Talamon-Fränkel in the ear secretion of 33 out of 43 cases examined by him; he also comments upon the remarkable fact that these exudations are practically never accompanied by perforation of the tympanic membrane. Zaufal[3] says there are ear-diseases "which run a strictly pneumonic course; they are ushered in with a chill, the temperature falls, and recovery takes place by crisis on the seventh or eighth day." This observation of Zaufal led Haug[4] and Brieger[5] to "assume a strictly pneumonic character and course for otitis due to diplococcus infection," or at least to point out the similarity "evidently existing in many particulars between genuine pneumonia and acute purulent otitis media." If they had read two lines further in Zaufal's article they would have seen that he considers it practically certain "that otitis due to streptococcus infection may run exactly the same course." When it is remembered that the diplococcus pneumoniæ is simply a pathogenic organism which does not produce pneumonia exclusively, and may give a general septic infection in no way distinguishable from that produced by other pyogenic organisms; and when, on the other hand, it is considered that diplococci are constant inhabitants of the upper air-passages in the healthy body, and can easily reach the middle ear and set up a suppuration if the condition of the mucous membrane is favorable, just like any other pathogenic organism that

[1] "Comptes rendus de la soc. de biolog.," 1890.
[2] "Jahrb. f. Kinderheilk.," XXXVII, p. 328 *et seq.*
[3] "Arch. f. Ohr.," XXXI, p. 184 *et seq.*
[4] "Die Krankheiten des Ohres," etc., p. 50.
[5] "Klin. Beiträge zur Ohrenheilk.," p. 68.

may be constantly present in the air-passages, it is well-nigh incredible that the mere fact of this organism playing a certain not thoroughly understood rôle in croupous pneumonia, and setting up a suppuration in the middle ear, should be utilized as a base on which to rear, with infinite art and ingenuity, the edifice of an entirely new disease, under the name of "pneumonic otitis media."

Wreden [1] maintains that disease of the ear may be caused by *atelectasis*, *bronchiectasis*, and *capillary bronchitis;* citing in explanation Lucae's observation that under normal conditions there is a regular pressure variation synchronous with the respiration. Believing, with Lucae, that the middle ear is in this way ventilated with every respiration, he concludes that the ventilation is insufficiently performed whenever the respiration is impaired, and consequently any disease which is attended with reduction of the respiratory function may be followed by disease in the ear.

We have already said enough on this subject to show that we consider these opinions as disposed of, but Wreden must nevertheless be given credit for having pointed out the frequency of ear anomalies observed at the autopsy in infants about a year old who died of pneumonia, presumably the catarrhal form. Later, Rasch [2] examined the bodies of 43 children dead of *bronchopneumonia*, and in 42 instances found inflammatory conditions in the ears, which in 30 consisted in middle-ear suppuration; while Ponfick,[3] in 10 out of 11 cases of uncomplicated pneumonia, found middle-ear suppuration at the autopsy, the ages ranging from one month to four years. In the absence of more convincing information, especially of a clinical nature, the question whether catarrhal pneumonia is the real cause of the suppuration must remain undecided. In the present state of our knowledge it seems more likely that the childish organism is predisposed to suppuration of the middle ear by any disease which seriously interferes with its nutrition, whether it be catarrhal pneumonia or any other affection. Later on we shall study the significance of intestinal affections in the production of ear diseases in young infants, and shall then learn that occasionally nutritive disturbance, by its weakening effect on the general resisting

[1] "Mon. f. Ohr.," 1868, p. 105 *et seq.*
[2] *Loc cit.*
[3] "Berl. klin. Wochen.," 1897, p. 852.

power of the infantile organism, is the predisposing cause of the aural complication.

The possibility of infection in the opposite direction deserves passing mention. Bronchitis and bronchopneumonia occasionally develop after purulent otitis media by aspiration of particles of pus and the contained bacilli which have found their way into the pharynx from the middle ear.

II. DISEASES OF THE CIRCULATORY SYSTEM.

1. DISEASES OF THE HEART AND BLOOD-VESSELS IN THEIR RELATION TO THE NOSE, PHARYNX, AND LARYNX.

Diseases of the heart and blood-vessels lead to—

1. Circulatory disturbances in the mucous membranes of the upper air-passages, producing hemorrhages, hyperemia, and congestive catarrh.

2. Motor disturbances by direct injury to the laryngeal nerves which are situated in their immediate neighborhood.

3. Pulsation of the large arterial trunks, when they are diseased, is transmitted to various portions of the upper air-passages.

4. Aneurysm of the aorta may lead to stenosis of the trachea or rupture into that tube.

Hemorrhages from the mucous membranes of the upper air-passages constitute a frequent concomitant of cardiac disease without compensation, and occur also, in consequence of the rise of arterial pressure, when compensation exists ; they are most common in venous stases due to failure of compensation in mitral disease and in aortic insufficiency. They are also observed in *arteriosclerosis*, and Edgren [1] reports the occurrence of epistaxis "during the presclerotic period, at a time when the only recognizable symptom is a heightened arterial pressure" ; he considers it, when occurring in elderly persons without apparent cause or after violent emotion, a symptom of incipient arteriosclerosis. The attacks soon cease to appear, even when they have been severe at one time. During the later stages of arteriosclerosis "the attacks of epistaxis appear to diminish in frequency, probably because of lowered blood pressure and lessened cardiac activity" (Edgren).

[1] "Die Arteriosklerose," Leipzig, 1898.

Epistaxis is the commonest form of bleeding from the mucous membranes; hemorrhages from the pharynx and larynx are rare. Although the usual, one might almost say the constant, seat of epistaxis is the spot known as locus Kieselbachii, on the cartilaginous portion of the septum,—recognized by the greater density of the vascular plexus,—the hemorrhages which occur in general circulatory disturbances often appear to originate in the lateral walls, and especially in the cavernous tissue. It is, unfortunately, impossible to locate the bleeding point while the hemorrhage continues, and even after the bleeding has stopped it is not always possible to determine its origin, on account of the hyperemic condition of the nasal mucous membrane and the presence of blood-clots. Hemorrhages have been reported from the veins at the base of the tongue, which sometimes become enormously engorged in conditions of passive hyperemia, and Compaired [1] mentions hemorrhage from the plexus on the glosso-epiglottidean fold in mitral insufficiency.

The hyperemia in course of time gives rise to *congestive catarrh*, involving the entire mucous membrane of the upper air-passages, and presenting the symptoms and clinical appearances seen in chronic rhinitis, pharyngitis, and laryngitis. The recognition of the symptoms of these forms of chronic catarrh is important, as it materially affects the treatment. Local measures are, of course, little adapted to effect a cure; painting with silver nitrate solution, which for some reason is such a favorite mode of treatment, is absolutely useless as long as constitutional treatment is neglected.

Passive edema in the larynx is a late complication, which does not develop in heart disease until failure of compensation has led to general edema; it therefore has no great value in diagnosis, as the local symptoms at this period are always overshadowed by the general phenomena.[2]

The *paralyses* which occur in the course of cardiac and vascular disease find their explanation in the proximity of the recurrent nerves to the great vessels in the mediastinum. The inferior laryngeal nerve is a branch of the pneumogas-

[1] "Ann. des mal. de l'oreille," 1896, p. 470.

[2] In "Arch. f. Laryng.," vol. VIII, No. 3, v. Sokolowski gives a description of the "morbid changes in the upper portion of the respiratory tract in the course of valvular disease."

tric, arising in the mediastinum, and, as its name—recurrens—implies, running back and upward to the larynx. The two nerves follow a different course, and therefore come into relation with different structures on the two sides of the body. The left nerve winds around the aorta and ascends along the posterior margin of the lateral wall of the trachea, in the groove between it and the esophagus, to reach the larynx; the right arises from the vagus at the level of the subclavian artery, winds around this vessel from before backward, and follows a course between the trachea and esophagus similar to that of its fellow.

The commonest causes of disturbances in the upper air-passages are found in dilatations of the great vessels, due to aneurysm. The most important are the *aneurysms of the aorta*, the symptoms of which require a detailed description. They consist in paralysis of the recurrent by direct injury to the nerve; transmitted pulsation of the larynx and trachea; tracheal stenosis from displacement of the wall of the trachea; and, lastly, rupture of the aneurysmal sac into the trachea.

Since the left recurrent nerve is in contact with the entire circumference of the arch of the aorta, it is affected by any aneurysm exerting pressure or traction on that structure. Traube [1] was the first to describe a paralysis of the recurrent due to aneurysm of the aorta, and since his day innumerable similar cases have been reported, so that paralysis of the left vocal cord has become one of the most important symptoms in the diagnosis of aneurysm of the aorta.

Paralysis of the recurrent nerve—a term by which, as will be more fully explained in treating of diseases of the nerves, is meant complete paralysis of all the muscles supplied by the inferior laryngeal nerve, the adductors as well as the abductors—is a typical symptom of aneurysm of the aorta, and, on account of the peculiar hoarseness it produces, rarely escapes the notice of either the doctor or the patient. It is quite different with the other form of paralysis of the recurrent, which affects only the cricoarytænoideus posticus, and exerts but little influence on either phonation or respiration, so that for several reasons it is not often observed in aneurysmal disease. It represents the

[1] "Deutsche Klinik," 1860, No. 41.

early stage of paralysis, and may be present when the aneurysm is beginning to develop, before any clinical symptoms have made their appearance. As this form of paralysis produces no functional disturbances, it escapes the notice of the physician, unless it is accidentally discovered in the course of a laryngoscopic examination.

It is owing to these two facts—the gradual, and at first painless, development of the aneurysm and the absence of symptoms in paralysis of the posticus—that the disease does not, as a rule, come under observation until it has made considerable progress, and the change from the median to the cadaveric position, which is the outward sign of paralysis of the recurrent, has taken place. Among other motor disturbances in the larynx in aneurysms of the aorta may be mentioned *laryngospastic attacks* and *periodic palsies of the vocal cords.* Löri and Grossmann have described certain laryngeal disturbances which are rarely observed as symptoms of incipient aneurysm of the aorta. Löri [1] says that the pressure of the aneurysm on the recurrent nerve in some cases provokes transient motor phenomena in the muscles of one-half of the larynx, which manifest themselves in difficult articulation ; in hoarseness, occurring at frequent intervals and without discoverable cause ; in sudden changes of the voice or of a single note ; and occasionally in spasm of the vocal cords. These phenomena, however, which are due to the irritation of very slight pressure, according to Löri, are replaced after a few days or weeks by paralysis of the entire half of the larynx from the increased pressure on the recurrent nerve.

In agreement with Löri, Grossmann explains similar phenomena observed by him as the effect of irritation by the gradually increasing pressure of the aneurysm on the nerves. His case [2] is remarkable from the fact that he was able to observe it more than a year. The patient came to be treated for frequent attacks of dyspnea of short duration, before there was any suspicion of aneurysm. After one of these attacks Grossmann observed a "paralysis of the left vocal cord," which disappeared on the following day. A few days later there was another attack of dyspnea, also accompanied by "total left-sided paralysis

[1] "Die durch Allgemeinerkrankung bewirkten anderweitigen Veränderungen," etc., p. 61.

[2] "Arch. f. Laryng.," vol. II, p. 254.

of the vocal cord." It is not quite clear from the description whether we have here a paralysis of the posticus or of the recurrent. One year later unmistakable clinical symptoms of aneurysm had developed, and, with the appearance of a total left-sided paralysis of the recurrent, the laryngospastic attacks ceased.

We have so far confined ourselves to the effects of pressure on the left inferior laryngeal nerve by an aneurysm of the aorta. The explanation of those cases, first described by Gerhardt [1] and Bäumler,[2] in which left-sided paralysis of the recurrent is combined with a similar paralysis on the right side, or in which there is right unilateral paralysis of the vocal cords, presents greater difficulties, as the course of the right recurrent nerve does not make the occurrence of such a condition appear probable. Among similar cases may be quoted Onodi's,[3] in which the right vocal cord was fixed in the cadaveric, and the left in the median position, and Cartaz's case, in which there was marked dyspnea and both vocal cords were seen in the median line, two or three millimeters apart, immovable, with concave edges. It is remarkable how often Löri [4] found the right nerve involved; he reports three cases of paralysis of the right half of the larynx and two cases of bilateral paralysis. Bäumler gives as an explanation of his case that the aneurysm produced overfilling, or even an aneurysmal dilatation, in the right subclavian artery, or that it pressed on the nerve from below at its origin from the pneumogastric. Another explanation appears to me to be suggested by the fact that unilateral paralysis of the pneumogastric is capable of producing bilateral disturbances of mobility. Semon,[5] and before him Löri,[6] gives the following explanation: A peripheral stimulus of the pneumogastric is transmitted through the *afferent* fibers of that nerve to the center in the medulla; from there it passes into the two motor nuclei of the vagus (Semon calls them the accessory nuclei), and thus gives rise to a bilateral disturbance of motility (Johnson's theory [7]).

Aneurysms of the aorta ultimately produce changes in

[1] "Virch. Arch.," XXVII, p. 75. [2] "Arch. f. klin. Med.," II, p. 550.
[3] "Semon's Centralbl.," X, p. 429.
[4] "Semon's Centralbl.," VIII, pp. 358 and 493. [5] *Loc. cit.*, p. 62.
[6] Heymann's "Handb. der Laryng.," I, p. 615.
[7] Semon quotes "Med. Chir. Trans.," vol. LVIII, 1875.

the trachea; *pulsating movements*, which may extend to the larynx; *tracheal stenoses* by compressing the walls; and, finally, *pressure ulcers* and *perforations*.

The arch of the aorta curves over the left bronchus from before backward, and lies close to the left anterior aspect of the trachea, just above the bifurcation, so that it occupies the obtuse angle formed by the trachea and left bronchus. Even under normal conditions a movement can be observed in the spur of the trachea in the laryngoscopic image, caused by the transmitted pulsation of the aorta. When the arch and descending limb of the aorta are dilated by an aneurysm and brought into closer contact with the trachea, the pulsation is communicated to the entire trachea, and can be observed even in the larynx. Oliver suggests bending the patient's head back, so as to draw the larynx upward, for the purpose of bringing out tracheal pulsation, while Cardarelli [1] observes the pulsation by the movements of Adam's apple with the patient's head bent back, and even pretends to be able to diagnose the seat of the aneurysm by the oblique direction of the pulsating movements.

Compression of the windpipe by an aneurysm in most cases produces a so-called scabbard-like stenosis of the trachea on the left side, with stenosis of the left bronchus. When the aneurysm is in the ascending limb, or in the arch, the pressure may in rare cases be exerted on the right side of the trachea and on the right bronchus. It is important to recognize these tracheal stenoses, as the respiratory embarrassment might otherwise be attributed to paralysis of the vocal cords which is usually present at the same time. Tracheotomy under such circumstances is, of course, useless; even the introduction of a cannula to the bifurcation, beyond the seat of the stenosis, gives only a temporary relief, because the pressure of the cannula very soon produces decubital ulcers in the trachea, through which rupture of the aneurysm takes place.

The rupture of an aneurysm into the trachea or bronchus is not a rare occurrence, but the mechanism has been variously explained by different anatomists. Eppinger [2] believes that the tracheal rings are forced apart by the wall of the aneurysm, and that rupture takes place through secondary aneurysms which form between the separated

[1] "Centralbl. f. inn. Med.," 1894, No. 42, p. 988.
[2] Klebs, "Handb. der pathol. Anatomie," VII, p. 270 *et seq.*

rings. He saw no proliferation of the cartilage or ulceration of the mucous membrane: "The edges around the seat of rupture were turned toward the interior of the trachea, and regularly sharp or delicately serrated and scaly, just as in true traumatic ruptures." Other authorities have described "conversion of the cartilage into detritus in consequence of compressing aneurysms, and atrophy of the cartilage by a process of fatty degeneration." [1] According to Selter,[2] who examined five cases, ulcers form in the mucous membrane as a result of the pressure, and subsequently lead to rupture of the aneurysms into the trachea or bronchus, so that the rupture is prepared from without.

In rare cases, paralysis of the vocal cords follows disease of other arterial trunks. Selter [2] saw an aneurysm of the innominate artery with paralysis of the right recurrent; E. Meyer [3] describes the same lesion in aneurysm of the right subclavian artery; in another case, marked pulsation in the pharynx was referred to aneurysmal dilatation of the carotid.

A *pericardial exudate* sometimes gives rise to paralysis of the left recurrent. Bäumler [4] first pointed out that the same condition can also produce paralysis of the right recurrent. "If the exudate is very abundant, and distends the pericardium as far as the jugular notch, the engorgement of the veins which meet at that point may exert direct or indirect pressure on the right recurrent." The case he quotes, which seems to me entirely convincing, has been called in question by Landgraf,[5] because the autopsy showed some slight syphilitic alterations in the larynx.

The paralysis attains its greatest intensity at the height of the exudative process, and subsides with the pericarditis. In this respect Landgraf's case is instructive: a pericardial effusion developed after articular rheumatism, and produced at first a paralysis of the posticus in the median position, which developed into paralysis of the recurrent in the course of the next two weeks, but the paralysis disappeared when the primary disease was removed.

Palpitation of the heart is one of the reflex neuroses, due

[1] Klebs. "Handb. der pathol. Anatomie," VII, p. 270 *et seq.*

[2] "Virch. Arch.," 133; also comp. D. Gerhardt, "Virch. Arch.," 123, p. 201.

[3] "Arch. f. Laryng.," II, p. 263.

[4] "Arch. f. klin. Med.," II, p. 550 *et seq.*

[5] "Charité Ann.," XIII.

to irritation in the nose. It occurs in chronic rhinitis with hypertrophy and polypus formation, and sometimes takes the paroxysmal form, analogous to sthenocardiac attacks and cardialgia. An interesting phenomenon, which has not as yet been satisfactorily explained, is sudden death from heart failure, which sometimes takes place a few days after extirpation of the larynx. Stork [1] attributes the phenomenon to injury of a depressomotor branch of the superior laryngeus, which is not constantly present; Grossmann [2] thinks it is caused by a central irritation of the superior laryngeal or of the vagus during the operation, while Toti [3] reports, without explaining, a case in which acceleration of the pulse rate to from 160 to 180 occurred thirty hours after an operation for the total extirpation of the larynx; and after twenty-four hours more of uninterrupted tachycardia the patient died of cardiac paralysis.

2. DISEASES OF THE HEART AND BLOOD-VESSELS IN THEIR RELATION TO THE EAR.

Tinnitus aurium is a frequent symptom of disease of the heart and blood-vessels and of anemia or hyperemia of the vascular systems within the ear. Our knowledge of these conditions is unfortunately very scanty, and we are hardly more advanced than was v. Tröltsch twenty years ago, when he wrote: "There is no doubt that tinnitus aurium is much oftener due to vascular murmurs than the profession has been inclined to believe up to the present time, as we are in the habit of attributing them chiefly to the influence of the nervous apparatus. It is often impossible to decide which of the two varieties is present, and simultaneous processes in both the circulatory and the nervous apparatus are probably of still more frequent occurrence."

Before proceeding to the discussion of pathologic changes, let us direct our attention for a moment to the normal conditions in which we do not observe any vascular murmurs. Since Weil [4] could hear the heart-sounds communicated to

[1] "Wien. med. Wochen.," 1888; and Alpiger, "Langenb. Arch.," xl.
[2] "Wien. med. Presse," 1892, Nos. 44–46.
[3] "Deutsche med. Wochen.," 1893, p. 87.
[4] "Die Auscultation der Arterien u. Venen," 1875.

the blood stream as vascular murmurs by auscultation of the carotid in the neck, it might be supposed that they could be equally well heard over the internal carotid where it passes through the canal in the petrous portion of the temporal bone. The solid bone which lodges the labyrinth is excellently adapted to conduct the sound to the internal ear, and the position of the carotid near the anterior wall of the tympanum would appear to render its perception very easy. The fact that the sound is not heard appears to be due to the venous plexus which surrounds the artery within the carotid canal, and acts like a cushion to arrest the pulsations and soften the sound.

The sinus of the jugular vein lies beneath the cavity of the tympanum ; and unless there are venous murmurs, there can not be any sound transmitted to the ear.

The ear itself is provided with two systems of blood-vessels—one in the middle ear and one in the internal ear. The former is composed of various branches derived from the external and internal carotids ; the latter belongs to the internal auditory artery, a branch of the basilar. To the investigations of Eichler [1] and Siebenmann [2] we owe our knowledge of the distribution of the capillaries in the neighborhood of Corti's organ. It was found that the membranes of Reissner and Corti, as well as that portion of the zona pectinata contained between the external pillar and the ligamentum spirale, are quite free from blood-vessels, and therefore the sensitive terminal apparatus of the auditory nerve is as far as possible removed from the influence of the vascular system.

It follows, therefore, that since, in spite of the proximity of the great vessels, the healthy ear does not perceive vascular murmurs, one of two pathologic possibilities must account for the occurrence of vascular noises : there must be disease either of the organ of hearing or of the vascular system.

In the former case the pathologic changes in the organ of hearing bring about more favorable conditions for the perception of the normal blood murmurs ; either the sound is more readily conducted on account of alterations in the

[1] "Die Wege des Blutstroms im menschl. Labyrinth," "Abhandl. der math. phys. Cl. der kgl. sächs. Gesellsch. der Wissensch.," vol. XVIII, No. 5, p. 327.

[2] See "Handb. der Anatomie," edited by v. Bardeleben, vol. V, part 2.

bone or the presence of an exudate, or the irritability of the auditory nerve is heightened, so that noises which before were below its range of hearing are now appreciated by the sensory end-organs. The quality of this kind of tinnitus aurium, which must be included under the general head of entotic vascular murmurs, is not as yet sufficiently known to make a classification into definite types possible. The different characters of an arterial and a venous murmur, as they have been described, and the interruption of the sound by compression of the respective artery or vein are not constant symptoms and can not be utilized in making a diagnosis. We shall return to this subject in another place.

The second group of subjective noises observed in diseases of the heart and blood-vessels are due to the transmission of abnormal vascular murmurs to the healthy ear. Among these we must distinguish those which originate in the heart and those which begin in the vessels.

To the former class belong the noises heard in valvular disease and in aneurysm, in which blowing, breathing, and hissing sounds are often heard in the ear and described as pulsating, hammering, or knocking noises. These descriptions are so common as to arouse the suspicion that the patient is describing a sensory perception of the arterial pulse, and not a true tinnitus aurium. Such a confusion of sensory perception of periodic movements with auditory impressions is much more probable than appears at first sight; it is often met with to an astonishing degree in testing with the tuning-fork. Just as the patient who is not used to observing accurately distinguishes with difficulty between the *vibrations* imparted to the entire head by a fork of low pitch and the *tone* of the fork transmitted to the ear over the craniotympanic conducting arc, so he may be misled by the sensation of the arterial pulse, and interpret it as an auditory impression, for we observe these hammering and knocking noises whenever the cardiac activity is heightened. Any one can "hear" the beating of his heart after physical exertion or mental emotion, but he can not say with certainty whether the impression is due to cardiac or to vascular murmurs. The theory that what is perceived by the patient in heart disease is not the valvular murmur, but rather the heightened arterial pulsation due to increased cardiac activity, finds further confirmation in the observation that these "entotic vascular murmurs" are complained of

particularly in aortic regurgitation with its rapid pulse, which produces an arterial pulsation that is perceptible even in the capillaries.

The murmurs which originate in the vessels themselves are produced by eddies in the blood stream, not by any special action of the vessel walls. The most important predisposing causes are the size of the lumen and the elasticity of the vessels.

It appears from reports of cases, some of which will be given later, that entotic vascular murmurs, whether of arterial or of venous origin, are observed with great frequency in aneurysm, in anemia and chlorosis, and in arteriosclerosis ; they occur as the result of circulatory disturbances in general plethora, in alcoholism, and after intoxications which are followed by a rise in blood pressure, or vasomotor paralysis, especially after the abuse of tobacco, and after full doses of quinin and salicylic acid. In this class belong the vasomotor disturbances with tinnitus aurium which occur in paralysis of the sympathetic, in connection with hyperemia of the skin ; they represent a symptom of Basedow's disease, which, according to Möbius, must now be regarded as an intoxication depending on the loss of the function of the thyroid gland, and not, as was formerly supposed, as a disease of the sympathetic system.

Finally, there are subjective noises which occur after *wounds of the head* in connection with partial loss of hearing and vertigo ; they are usually attributed to vasomotor irritation. As these symptoms are usually observed only in cases of accidents, there is a natural tendency to ascribe them to traumatic hysteria and neurasthenia. This is the view adopted by Schwartze some time ago ; but Müller,[1] in a recent communication from Trautmann's clinic, pointed out that a wound of the head may give rise to irritation of the vasomotor center, manifesting itself first in contraction and later in relaxation and paralysis of the muscular walls of the blood-vessels ; this may in turn be followed by extravasations and permanent functional disturbances which explain the subjective symptoms complained of by the patient. The tinnitus aurium in this case is, therefore, to be regarded as the result of hyperemia manifesting itself at first in hyperemia of the tympanic membrane and

[1] "Deutsche med. Wochen.," 1898, No. 31.

external auditory meatus, which later may be replaced by cloudiness of the membrane.

The investigations in arterial auscultation by Weil [1] and v. Frey [2] show that the blood-vessels give forth a peculiar note, rarely heard in healthy individuals, but frequently in fever patients, in anemia and chlorosis, and in aneurysm; on the other hand, according to Weil's observations on the femoral artery, the tone was persistently absent in conditions of high arterial tension from atheromatosis and nephritis with hypertrophy of the heart. In the former case the results of auscultation coincide with the subjective ear symptoms, while in the latter the frequent occurrence of entotic vascular murmurs in arteriosclerosis is in marked contradiction to them. But we find an explanation for the occurrence of tinnitus aurium in atheromatosis in the investigations of Nolet,[3] who found that murmurs in the vessels may be caused by sudden changes in the pressure and velocity of the blood wave, such as are produced by changes in the lumen of the vessel. These conditions are most marked in arteriosclerosis when there are aneurysmal dilatations in the vessels. The behavior of the blood-vessels of the ear in arteriosclerosis has, unfortunately, never been examined anatomically, but it is safe to say that the production of entotic murmurs depends on the extent of atheromatous change and the presence of miliary aneurysmal dilatations; a unilateral tinnitus aurium, therefore, does not necessarily exclude an atheromatous origin, but merely suggests the existence of a local form. Stacke[4] reports a case characterized by the perception of marked subjective tones, high in pitch, combined with central deafness of the right ear; he explains the unilateral character of the symptoms by the existence of a circumscribed atheromatosis of the vessels in the right side of the neck.

Being convinced of the frequency of tinnitus aurium as a concomitant of arteriosclerosis, I examined for this symptom the 124 case histories of arteriosclerotic patients reported by Edgren,[5] but to my astonishment I found such complaints in only three of the histories, although Edgren himself remarks further on (p. 207) that vertigo and

[1] "Auscultation der Arterien u. Venen," 1875.
[2] v. Frey, "Die Untersuchung des Pulses," 1892, p. 6 *et seq.*
[3] "Arch. d. Heilkunde," 1871. [4] "Arch. f. Ohr.," XX, p. 286.
[5] "Arteriosklerose," Leipzig, 1898.

tinnitus aurium are complained of early in the disease by many patients. His interpretation of these complaints differs somewhat from my own views ; he finds the cause of the noises "in the brain," and attributes them simply to increased arterial tension, without any material alterations in the brain itself.

I shall now proceed to quote a few cases of subjective noises in the ear. Moos [1] reports a case in which the noises were very loud and compared by the patient to the noise of machinery and railroad trains ; at the autopsy the sinus of the jugular vein was found abnormally dilated. Wagenhäuser [2] attributes a case of marked tinnitus aurium, aggravated by cough and demonstrable objectively with the auscultatory tube, to an aneurysmal dilatation of the internal carotid ; but as the patient, a girl of nineteen, presented besides a marked emphysematous habit, a large goiter, and a cyanotic appearance, his explanation is open to criticism. Brandeis [3] regarded a noise which was heard in a disease of the upper cervical vertebræ as a vascular murmur emanating from a dilated vertebral artery. The literature contains many cases of aneurysmal dilatation in various vascular systems which produced subjective ear noises. Among the external vessels of the head the region of the temporal, occipital, and posterior auricular arteries furnishes examples quoted by Chimani [4] and Herzog. [5] Subjective and objective noises in the head may be of great significance in the diagnosis of aneurysm at the base of the brain. In the case of a woman who suddenly began to complain of tinnitus aurium and impaired hearing and lost consciousness, Varrentrapp [6] found at the autopsy a ruptured aneurysm of the basilar artery. Lebert,[7] in his studies on aneurysm of the cerebral vessels, calls attention to the frequency of tinnitus aurium as a symptom of aneurysm of the middle cerebral and basilar arteries ; in the case of the latter it may have great diagnostic value as an early symptom. Deafness has often been observed in combination with the subjective noises ; sometimes it comes on

[1] "Arch. f. Augen- u. Ohrenheilk.," vol. IV.
[2] "Arch. f. Ohr.," XIX, p. 62.
[3] "Zeitschr. f. Ohr.," vol. XI.
[4] "Arch. f. Ohr.," VIII.
[5] "Mon. f. Ohr.," 1881, Nos. 8 and 9 ; with review of cases reported up to date.
[6] "Arch. d. Heilkunde," 1865.
[7] "Berlin. klin. Wochen.," 1866, pp. 251, 282.

suddenly, and must be explained partly by the obliteration of the arteries supplying the ear, and partly as the result of pressure on the auditory nerves. Oppenheim [1] was able to auscult a loud pulsating murmur over the left half of the skull, which, because of a coexisting ocular disturbance, he referred to aneurysm of the posterior communicating artery; but there is no record in the history that the patient had been aware of the murmur. Hyrtl [2] contributes the observation that the artery of the stapes is sometimes very large, and in that case is likely to give rise to vascular murmurs.

When the *character of the entotic vascular murmurs* is examined, it is found that the difference between arterial and venous murmurs has been very differently described. The arterial murmurs are said to have a distinct pulsating character, to be synchronous with the apex-beat, and to manifest themselves "as a series of buzzing or pounding noises in the ear or in the head" (Kayser[3]), whereas the venous murmurs are breathing or blowing in quality, and continuous. As we must depend for a description of the murmurs on the statements of the patient,—for even when an aneurysmal bruit can be heard objectively we have no means of judging whether the patient hears the noise in the same way,—it is easily understood why the descriptions vary so widely. The patient naturally chooses a comparison from his surroundings or from among the sounds he has become familiar with in his calling, so that the murmurs have been compared to the rush of water over a dam, the rustling of leaves in the forest, the noise of machinery and railroad trains, the hiss of boiling water, the chirping of a cricket, etc.

A few examples are given to show that even the general character of the arterial and venous murmurs, as just described, does not apply in every case. Kayser lays down the rule that arterial hyperemia, like the inhalation of amyl-nitrite, produces low-pitched, buzzing sounds, while anemia, like syncope, gives rise to high, resonant tones. According to v. Tröltsch, the predominant characteristic of the noises in anemia and chlorosis is hissing and blowing. According to Stacke, in arteriosclerosis the subjective noises are high in pitch; and it is worthy of remark that although of arterial

[1] "Berlin. klin. Wochen.," 1896, p. 402.
[2] Quoted by Urbantschitsch, "Schwartze's Handb.," vol. I, p. 413.
[3] Bresgen's collection II, part 6, p. 28.

origin, the sounds are not intermittent in character. Moos[1] points out that they are aggravated by anything which tends to stimulate the circulation. In his case—mentioned in another place—he ascribes the subjective noises to a marked dilatation of the sinus of the internal jugular vein. The noises which the patient compared to the din of machinery and railroad trains were so intense that they drove him to commit suicide. The interpretation of this case is open to criticism, as there evidently existed a psychosis. The noises caused by heart disease and aneurysm are usually described as intermittent and buzzing or soughing in character.

The differential diagnosis between entotic vascular murmurs and simple noises in the ear can be established in some cases by compressing the corresponding vascular trunks—that is, the external and internal carotids, the vertebral artery, and the internal jugular vein—which are concerned in the blood supply of the ear. The effect produced by compressing the blood-vessel will vary according as the vascular murmur is arterial or venous in character, and emanates from the distribution of the carotids in the tympanum or from the branches of the vertebral artery in the internal ear. Schwartze [2] mentions the disappearance of pulsating murmurs after compression of the carotid, and suggests "ligation of the carotid for the cure of aneurysm" to remove the murmurs. In Wagenhäuser's case, where aneurysm of the internal carotid was suspected, the vascular murmur was diminished by pressure on that vessel; in Oppenheimer's case of aneurysm of the posterior communicating artery, in which the murmur was heard only objectively, compression of the carotid had no effect. Von Tröltsch [3] quotes Türck as saying that pressure on the first cervical vertebra alters and usually diminishes the murmur momentarily, and, similarly, Dundas Grant[4] recommends compression of the vertebral artery in cases of vascular murmurs, so as to relieve the tension in the distribution of the basilar artery, of which the internal auditory is a branch.

Although the general impression prevails that pressure

[1] "Schwartze's Handb.," vol. I, p. 535.
[2] "Die chir. Krankh. des Ohres," p. 170.
[3] "Lehrb.," 7th edition, p. 606.
[4] Quoted by Brieger, "Klin. Beitr.," p. 139.

on the internal jugular vein—as, for instance, by struma or a tight collar—produces tinnitus aurium, Boudet[1] maintains that the noises can be suppressed by compression of the vein.

On the subject of *embolic disease of the ear in endocarditis* we have the investigations of Trautmann,[2] which confirm the extreme rarity of its occurrence and the absence of marked symptoms, at least in thrombosis of any of the smaller vessels. Embolism of the basilar or of the internal auditory artery may give rise to sudden deafness, as happened in a case of Friedreich's ;[3] but emboli in the smaller arterial branches of the middle ear do not necessarily cause any functional disturbance.

Trautmann's anatomic investigations on the cadaver convinced him that *embolic processes* are more apt to occur in the tympanum than in the internal ear, because the arterial path from the posterior auricular to the stylomastoid is straighter than that which leads from the tortuous vertebral artery to the basilar and internal auditory. It appears from Trautmann's observations that of thirteen cases of endocarditis four showed petechial hemorrhages in the tympanic membrane and the mucous membrane of the middle ear ; but his findings can not be utilized for ear diseases following simple endocarditis without a reservation, as most of his patients were cases of ulcerative endocarditis and general sepsis, showing septic embolism of cutaneous vessels with roseola-like macules and petechial hemorrhages. The changes in the ear may be regarded as analogous with the latter, and caused, not by endocarditis in general, but by the sepsis present in these cases. Habermann[4] recently reported a case of rather sudden deafness of the right ear, in which there was double mitral disease with endocarditis.

The prognosis of deafness after embolism of the internal auditory artery is unfavorable as to recovery of hearing, which differentiates this form from that due to hemorrhage into the central auditory tract, which usually ends in recovery by absorption.

[1] Quoted by Urbantschitsch from "Henle's Jahresber.," 1862, p. 520.
[2] "Arch. f. Ohr.," XIV, p. 73.
[3] Moos, "Wien. med. Wochen.." 1863, p. 661.
[4] "Verhandl. der D. otol. Gesellsch.," 1898, p. 90.

III. DISEASES OF THE DIGESTIVE SYSTEM.

1. DISEASES OF THE DIGESTIVE SYSTEM IN THEIR RELATION TO THE UPPER AIR-PASSAGES.

DISEASES AND CHANGES IN FORM OF THE ORAL CAVITY IN DISTURBANCES OF NASAL RESPIRATION.

Morbid changes in the oral mucous membrane and changes in the shape of the oral cavity result from obstruction of the nasal chambers; the etiology of the latter and its effect on the respiratory passages has already been fully discussed. The inspiratory air current, in passing through the mouth, exerts a cooling and desiccating influence on the mucous membrane, giving rise to a subjective feeling of dryness in the mouth and throat, and, from the deposition of dust, to a stale, disagreeable taste and general anorexia. It seems probable that mouth-breathers are more exposed to catarrhal affections of the gums and of the mucous membrane covering the tongue and oral cavity on account of the greater facility of direct infection, but the supposition has never been proved, any more than the statement that they are more disposed to inflammation of the tonsils.

Since Moldenhauer and Bloch, among others, called attention to the changes produced in the shape of the upper maxilla by obstruction of the nasal respiration, the subject was carefully investigated by Körner [1] and by his disciple Waldon,[2] and their statements are confirmed by the observations of others. Körner divides the malformations of the jaw into those which occur before the period of second dentition and those which are produced if there is nasal obstruction while that process is going on.

[1] "Untersuchungen über Wachsthumsstörungen und Missgestaltung des Oberkiefers und des Nasengerüstes in Folge von Behinderung der Nasenathmung." Leipzig, F. C. W. Vogel, 1891.

[2] "Arch. f. Lar. u. Rhin.," vol. III, p. 233 *et seq.*

The first consists in a "dome-like elevation" of the palate, the highest point of which corresponds to the anterior portion of the roof of the mouth, the posterior surface of the median portion of the alveolar process rising almost perpendicularly behind the incisors. The curve of the alveolar border, which in normal impressions is usually seen to correspond to a semicircle, takes the form of an ellipse.

When the deformity develops during the period of second dentition, there is, in addition to these changes, a marked upward growth of the superior maxilla in the sagittal axis, and a corresponding diminution in the transverse diameter, so that the jaw appears both high and narrow. The teeth, which had not been affected before, also show the effect of the deformity in the position of the central incisors, which, owing to the lateral approximation of the alveolar processes and their meeting in an acute angle in the median line, are placed with their posterior surfaces facing each other. And as, in consequence of the excessive lengthening of the jaw, the anterior alveolar border is pushed forward and loses its perpendicular position, the incisors necessarily take the same direction, and usually project beyond the lower teeth, reminding one of a rodent. In addition to all these changes, there is a general hypoplasia of the superior maxilla, which is regarded as a kind of arrested development due to the respiratory inactivity of the nose. The interior of the nose is also undeveloped, and this explains the upward growth of the palate. The lateral contraction of the palate is explained, after Körner, by "the pressure exerted on the sides of the jaw by the stretching of the cheeks when the mouth is open"; this explanation seems plausible, since it is generally accepted that the mouth is at rest when closed, and the act of opening it, which in mouth-breathers becomes habitual, is associated with contraction of the muscles about the jaw.

Although the lower jaw is equally subjected to the lateral pressure of the contracted muscles (which produce the approximation and protrusion of the alveolar process of the upper jaw), a similar malformation can not result, because a counterpressure is maintained from within by the tongue, which fixes the rami of the jaw in their normal positions. Hence the lower jaw does not, like the upper, suffer any alterations when nasal respiration is obstructed.

The habit of keeping the mouth open results in atrophy

of the orbicularis oris, which shows itself in the diminished width of the lips and shortness of the upper lip, so that the lower half of the teeth are not covered.

DISEASES OF THE DIGESTIVE ORGANS IN RELATION TO THE NOSE, THROAT, AND LARYNX.

Diseases of the teeth play no inconsiderable part in the pathology of the antrum of Highmore and of the nose; the ulceration may spread through the alveolar process to the mucous membranes of these cavities, a dental cyst may simulate an empyema, or a tooth may even develop in the antrum of Highmore or in the nose.

While it must be admitted that diseases of the teeth occupy a prominent place in the etiology of suppurative processes in the antrum, it would be a great mistake to fall under the influence of the dentists, who have claimed the pathology of the tributary cavities as their own province, and neglect other sources of infection for the nose. It is true that many cases of empyema of the antrum are due to infection derived from a carious tooth or to the encroachment of a dental cyst; but if every suppuration of the antrum is to be referred to disease of the teeth, how shall we explain the inflammations which occur in the frontal sinuses quite as frequently as in any of the other accessory cavities?

The danger to the antrum of infection from a decaying tooth varies with the individual tooth, the anatomic relations of the alveolar process, and the size of the accessory cavity concerned.

The lumen of the antrum may be conveniently described as representing a pyramid: the base corresponds to the lateral wall of the nose, the apex lies in the zygomatic or malar process, and the three sides are formed by the inner aspects of the facial, orbital, and pterygopalatine or zygomatic surfaces of the superior maxilla. The junction of the facial and nasal walls of the cavity comes into close relation with the alveolar process, but nearer the median line the sockets are separated from the floor of the cavity by a thicker ridge of bone. The longer the alveolar process, as roughly determined by the elevation of the roof of the mouth, the thicker the mass of bone which separates the roots of the teeth from the antrum, and the less

prominent their outlines on the inner surface of the cavity. These relations are, of course, variable, and the possibility of a morbid process spreading from the teeth to the antrum depends on whether the roots of the teeth are separated from the cavity by a thick layer of bone or only by a slender lamella and the epithelial lining of the antrum.

It may be laid down as a rule, independent of these varying anatomic relations, that certain teeth are always nearest the cavity, and therefore most dangerous to the antrum if they become diseased ; while, on the other hand, they also offer the readiest means of access to the antrum for therapeutic purposes. The floor of the antrum is deepest over the second bicuspid and first molar ; hence, whatever the thickness of the intervening bone at other points, these two teeth always lie nearest the cavity, and constitute the point of election for attacking the maxillary sinus through the alveolar process.

On the subject of periodontal cysts and their extension to the antrum of Highmore there is a paper by Kunert[1] in which he points out the diagnostic points between such a cyst and true empyema of the antrum. I am willing to admit that protrusion of the facial and orbital plates and of the hard palate is characteristic of cysts ; but in bulging of the outer wall of the nose, accompanied by a flow of pus from the middle meatus,—symptoms referred by Kunert to the spontaneous opening of a cyst,—I believe the rhinologists will be inclined to exclude any cystic condition from the etiology. Kunert betrays his imperfect acquaintance with rhinology when in the diagnosis of a true empyema of the antrum he utterly ignores the significance of granulations and polypi in the middle meatus, coupled with disease of other adjoining cavities.

The presence of a tooth in the inferior meatus on the floor of the nose or in the antrum admits of two explanations : either it is an inverted tooth or it is the product of a dental papilla which wandered into the nasal cavity before closure of the palatal cleft had taken place. Sometimes a foreign body lying loose on the floor of the nose, and covered or surrounded with swollen mucous membrane, is removed from the nose, and, to the surprise of the surgeon, turns out to be a fully or only partly developed tooth,

[1] "Arch. f. Laryng.," VII, p. 34.

which must have been there for years without causing any symptoms.

The theory which formerly prevailed, that spasm of the vocal cords in children is due to difficult dentition, has been disproved. As will be shown later on, in the section on rachitis, the phenomenon must be regarded as a symptom of the general impairment of nutrition, more particularly of the rachitic habit.

The diseases of the palate and of the oral pharynx will be found fully treated in the special text-books devoted to them, and need not be discussed here.

Diseases of the *esophagus*, in the form of tumors, diverticula, and peri-esophageal abscess, have their effect on the upper air-passages whenever the larynx and trachea become involved in the morbid process, or whenever the tumor produces paralysis by pressure on the laryngeal nerves. Malignant tumors originating in the highest portion of the esophagus, at the level of the cricoid cartilage, are prone to spread into the interior of the larynx, and it is often difficult to decide, by the laryngoscopic image, whether the primary seat of the tumor is in the larynx or in the esophagus. Whenever there are distinct signs of carcinomatous changes in the interior of the larynx, and a mass suddenly makes its appearance in the pyriform sinus, or, as is sometimes observed, pushes its way into the lumen of the larynx over the interarytenoid notch, it may be said with certainty that the tumor has invaded the esophagus. In operating on such cases it must be remembered that the process has probably attained such dimensions that there is no possibility of a radical cure without extensive resection of the esophagus and pharynx.

The possibility of tumor or dislocation of the esophagus producing paralysis of the vocal cords follows logically from the course of the recurrent nerves in the groove between the trachea and esophagus, which has been sufficiently described in another place.

Dyspepsia is often found associated with atrophic fetid rhinitis and pharyngitis and with abscess in the cavities adjoining the nose, obviously because the pus which enters the pharynx is often swallowed. It would be well worth while to examine these relations more closely from a clinical standpoint, for, as far as my experience goes, this cause for chronic gastric catarrh has so far barely received a passing mention. When complaints of failing appetite

and bad digestion are constantly heard in cases of ozena, where the cavities of the nose are enormously enlarged and its walls covered with crusts, where the pharynx and posterior pharyngeal wall is filled with offensive discolored masses of secretion, it seems but natural to attempt to establish a causal relation between the two conditions. A secondary chronic gastritis is readily explained either by the anomalies of smell and taste which result from the ozena and manifest themselves in paresthesiæ and anesthesiæ, destroying the appetite and causing a bad taste in the mouth, or directly by the irritation of the decomposing secretions in the stomach.

The American literature [1] contains a few observations on the significance of dyspepsia in the etiology of rhinopharyngeal catarrh. Beverley Robinson's remarks on this subject are worth quoting: "Dyspepsia," this writer says, "increases an already existing pharyngeal catarrh, because the eructations of gas act as an irritant, and the acid matters, which contain large quantities of butyric acid and similar substances, tend to aggravate the condition."

In regard to spasm of the vocal cords in infants, which is said to be caused by defective nutrition of the sensory nerve-endings of the vagus in the stomach, there is a discussion by Rehn,[2] which will be referred to again in connection with rachitis.

The theory that cough may be produced by reflex irritation of the pneumogastric in the stomach was formerly accepted by physicians, and even now enjoys a wide recognition among the laity, as we know by the generally accepted term "stomach-cough."

The symptom has now entirely disappeared from the literature, for the possibility of such reflex irritation has been denied on theoretic grounds (Nothnagel, Naunyn); nor does the literature furnish any cases which can be accepted as proving it absolutely. Even the case reported by Bull,[3] in his paper on stomach-cough, which is supposed to be a clinical observation of stomach-cough of reflex origin from irritation of the gastric walls, does not convince me, as the paroxysms, which could be brought on by pressure on the epigastrium, as well as the entire course

[1] Ref. "Semon's Centralbl.," VI, p. 83; X, p. 349.
[2] "Berlin. klin. Wochen.," 1896, No. 33.
[3] "Deutsches Arch. f. klin. Med.," vol. XLI.

of the disease, with its repeated relapses, appear to me to have a distinctly hysteric character.

When the intestines are in a state of irritation from the presence of parasites, reflex tickling sensations and a desire to sneeze are often felt in the nose, especially when the irritation is in the rectum. These conditions are usually due to excessive acidity of the urine, although it is admitted that they may be caused by the presence of seat-worms and tape-worms. The statement is occasionally met with that spasm of the glottis may be due to reflex irritation of worms.

In *cirrhosis of the liver*, owing to the impaired nutrition of the vessel walls, or as the result of a primary hypertrophy of the heart, hemorrhages occur in the mucous membranes, sometimes severe enough to constitute epistaxis, but usually merely in the form of ecchymoses in the larynx and post-nasal cavity. Cases of genuine laryngeal hemorrhage are very rare; Dreyfuss [1] lately published two cases, the etiology of which, however, was somewhat obscured by the presence of other laryngeal disease. These hemorrhages and varicose conditions are easily explained, as are hemorrhages and varicose veins in the esophagus, by the free anastomosis existing between the laryngeal veins and the tributaries of the inferior and superior thyroid, and, through them, with the peri-esophageal veins which belong to the portal system.

The *icteric* hue manifests itself in the mucous membranes by a lemon-yellow color, just as in the epidermis. In the larynx it is most marked in the valleculæ, above the epiglottis, and on the vocal processes. Paresthesiæ in the throat have been described, exciting cough and hawking. A few cases of paralysis of the vocal cords have been reported in jaundice with fever. Gerhardt [2] and Hertel [3] describe a paralysis of the adductors, with gaping of the glottis during phonation, and moderate injection of the vocal cords; the paresis diminished as the jaundice disappeared, and phonation was gradually restored. In these cases there may have been an intoxication of the nerves, owing to their absorbing the cholates, or it may be that paralysis occurs only in the infectious form of jaundice, known as Weil's disease, for the course of both Gerhardt's and Her-

[1] "Münch. med. Wochen.," 1898, No. 32.
[2] "Die med. Wochen.," 1887, p. 325.
[3] "Charité Ann.," 1891, XVI.

tel's cases strongly suggest that disease. M. Schmidt [1] calls attention to the paroxysmal cough sometimes excited by the reflex irritation of gall-stones, and mentions Cahn's case of vasomotor coryza (hydrorrhea nasalis) directly caused by *hepatic colic*, and another in which there was a causal relation between vasomotor coryza and round ulcer of the stomach.

Finally, *cholera asiatica* and severe cases of ordinary *cholera* may give rise to various disturbances in the upper air-passages. The most familiar of these is the aphonia, or *vox cholerica*, which accompanies the attack of cholera; it is usually attributed to weakness of the muscular tissues which are deprived of the necessary moisture. Matterstock [2] made a series of laryngoscopic examinations in the Würzburger Klinik during a cholera epidemic. It appears from his investigations that the interior of the larynx becomes cyanotic, and the vocal cords discolored and moderately injected. The most conspicuous change was a marked prominence of the vocal processes, the vocal cords being very much hollowed out, so as to present the shape of a sickle. Wide gaping of the glottis during phonation accounted for the aphonia, which was not constantly present, the patient regaining his voice temporarily under the influence of excitement or after the application of the faradic current. Matterstock rejects the foregoing interpretation, which is the one generally accepted, and refers the paresis to nervous influences; the aphonia, according to him, is not dependent on the excessive loss of fluid, so that one might regard the vox cholerica as a toxic paralysis, analogous to those which occur in the course of other infectious diseases.

The croupous and diphtheric inflammations of the pharyngeal and laryngeal mucous membranes in cholera are interpreted by Liebermeister [3] as superficial necroses, due to the profound disturbance of nutrition and circulation, comparable to similar complications in typhoid fever, variola, and puerperal fever.

[1] "Die Krankh. der ob. Luftwege," 2d ed., p. 749.

[2] "Berl. klin. Wochen.," 1874.

[3] "Die Cholera," in Nothnagel's "Spec. Path. u. Therap.," vol. IV, 1, p. 68.

2. DIGESTIVE SYSTEM AND DISEASES OF THE EAR.

The influence of disease of the intestinal canal on the ear is inconsiderable. Moos and Haug refer to auditory disturbances said to have been observed by Ménière [1] in gastric disease, and Haug mentions one or two other unimportant cases, but both devote a great deal of attention to diseases of the teeth as the cause of aural disturbances.

Of all the complications presently to be mentioned there is only one—neuralgia localized in the ear after caries of the teeth—that appears to me to be definitely proved. As for the exudative otitis media in diseases of the teeth, which is said to result from reflex irritation of the dental branches of the trigeminus,—which, as will be explained in another place, exercises a trophic influence on the mucous membrane of the middle ear,—I do not consider that the etiologic relationship has been clearly established.

It happens every day that a patient comes to the doctor complaining of earache, and the cause of this alleged "earache" is found in a carious tooth; or a patient with acute otitis media describes the pain as radiating to the molars. It is, no doubt, this radiation of the pain in earache that is responsible for the popular belief that toothache can be relieved by dropping warm oil into the external auditory meatus, and induces many women of the lower classes to put cotton in their ears for the same purpose. It is not at all surprising that the people at large should harbor the superstition that a running ear may be caused by the process of first or second dentition, when we remember the layman's talent for confusing cause and effect and his remarkable ingenuity in interpreting reflex conditions, before which the inventiveness of the most ingenious discoverer of reflex neuroses sinks into insignificance; but that such views should still prevail in medical circles [2] is simply incomprehensible. It is indeed difficult to conceive how a purulent otitis media could be the result of the physiologic process of dentition, except on the very improbable hypothesis that the inflammatory irritation of the gums was communi-

[1] "Rev. mens. de lar.," etc., 1886, No. 6.

[2] For instance, quoted by Moos: Burnett, "Am. Journ. of Otol.," vol. II, part IV, p. 285.

cated to the middle ear by way of the postnasal space and the Eustachian tube. For similar reasons I am inclined to deny any connection between purulent otitis media and caries of the teeth, and the cases published in support of the theory do not appear to me to bear the scrutiny of closer inspection. Thus, a woman has a tooth filled, and during the following night is taken with vertigo and headache, her hearing is impaired, and she has tinnitus aurium. A week later, suffering with acute middle-ear catarrh in process of regeneration, she comes under the care of an ear specialist, who concludes that the aural inflammation is the result of filling the carious tooth. But it is much more likely that the patient originally referred the pain of an acute otitis to a tooth, and as she happened to have a carious tooth at the time, she had it filled, without, of course, arresting the course of the inflammation ; when, a week later, the otologist found only the remains of an otitis, he hastily concluded, without going into the case very critically, that the carious tooth was the primary cause. A similar case was observed by Blau ; [1] he, however, was too critical to admit an etiologic connection between the dental and the aural conditions without a reservation. Haug,[2] on the other hand, attributes a case of hemorrhagic exudation into the tympanic cavity, with ecchymoses in the external auditory meatus, to pulpitis of a molar tooth. The spontaneous cure of the aural affection within sixteen days after the tooth was extracted does not confirm the diagnosis, as any uncomplicated acute hemorrhagic inflammation would have subsided just as rapidly. Nor is there any better proof in a case described by Schwartze[3] as "acute purulent otitis media with caries of the mastoid process resulting from caries of a second molar."

The *ingestion of food* may be seriously interfered with if the ear disease involves the articulation of the lower jaw, and mastication, or even opening the mouth, becomes painful or impossible. Such disturbances occur most commonly in otitis externa, with furuncle on the anterior wall of the meatus, and infiltration of the parts about the joint. Extensive caries of the temporal bone and malignant tumors

[1] "Arch. f. Ohr.," XXIII, p. 12.
[2] "Verhandl. der D. otol. Gesellsch.," 1895, p. 41.
[3] "Zeitschr. f. Ohr.," XXIII.

in the external or middle ear may destroy some of the tissues entering into the construction of the joint.

Disturbances of the sense of taste due to ear disease will be discussed in the section devoted to nervous diseases.

In persons afflicted with chronic purulent otitis media the trickling of pus through the Eustachian tubes into the pharynx (according to Itard [1]) sometimes produces nausea and vomiting, with bad taste in the mouth and indigestion. But it is chiefly during *infancy and early childhood* that the connection between *diseases of the gastro-intestinal canal* and inflammations and suppurations in the middle ear is noticeable. The striking coincidence of digestive disturbances and running of the ear, and the frequent presence of pus and inflammatory exudate in the ears of children who have died of enteritis, leave no room for doubt that we have to deal with a deeper etiologic relationship and not merely with an accidental coincidence.

It has taken a long time to arrive at a clear understanding of the nature of this connection ; up to a very recent date the most extravagant suppositions were entertained regarding the normal condition of the middle ear in the new-born and during the first months of the infant's life, and, in consequence, widely divergent interpretations were suggested for cases in which the autopsy revealed the presence of a mucopurulent secretion, with swelling and injection of the mucous membrane of the middle ear. The process of involution which takes place in the middle ear of the new-born, consisting in resorption of the so-called fetal pad of mucous membrane or mucoid embryonic connective tissue which fills the cavities of the middle ear during intra-uterine life, led certain authors to explain the presence of mucopus in the middle ear of infantile cadavers as a physiologic formation due to a persistence of the embryonic tissue.[2] This confusion of ideas continued until Hartmann [3] instituted his first systematic investigations on the cadavers of infants. The confusion was aggravated by the current belief that the diagnosis of diseases of the ear and the interpretation of the otoscopic image in infants were based on an entirely different set of

[1] Quoted by Urbantschitsch, "Lehrb.," 2d ed., p. 251.
[2] For the literature see Aschoff, "Zeitschr. f. Ohr.," XXXI, p. 345.
[3] "Deutsche med. Wochen.," 1894, No. 26.

principles, and offered peculiar difficulties, as compared with similar conditions in adults.

Hartmann repeatedly pointed out the significance of purulent otitis media in the nutritive disturbances of infants; then Ponfick [1] contributed his evidence to the elucidation of the subject, and Goeppert [2] showed that purulent otitis media is often a sequel of intestinal diseases in infants, which up to that time had been practically disregarded. We have already seen that the interdependence of lung and ear diseases has been known and studied for some time, and now Goeppert finds that the percentage of ear complications is much higher among children suffering from intestinal troubles than it is among those admitted for other diseases; 12% of the former and 39% of the latter class were found to have healthy ears. As to the mode of origin of ear diseases in gastro-intestinal affections, it is just as difficult to explain as it is to decide in cases of long standing whether the ear or the intestinal tract is the primary seat of disease. The question may, perhaps, best be answered by referring to what has been said in connection with purulent otitis occurring in the course of lung diseases: the power of the organism to resist infection having been weakened by disease, the infant is more prone to suppurative processes in the tympanic cavity; and in the same way marasmus must be regarded as a frequent cause of disease in the ear. Goeppert's theory, that infection during an intestinal disease occurs solely through the entrance of vomited matter into the middle ear by way of the tubes, can hardly be accepted in all cases.

Secondary nutritive disturbances play an important rôle in primary ear diseases in the case of infants. A regular digestion and a uniform increase in the body-weight afford the best criterion of an infant's health, for its digestive organs are so sensitive that the slightest local or general disturbance may suffice to upset its stomach. If there is a purulent focus anywhere in the body, auto-intoxication will be much more likely to result from the absorption of the products of metabolism in the infant than in the adult, and will show itself chiefly in the organs which are functionally the most important: that is, it will be followed by indigestion and a falling-off in weight. Thus, there is danger of

[1] Ponfick, "Berlin. klin. Wochen.," 1897, No. 38.
[2] Goeppert, "Jahrb. f. Kinderheilk.," vol. XLV, p. 1.

auto-infection whenever the secretions are retained in the middle ear in purulent otitis media, because there is no perforation, or only an insufficient one, in the tympanic membrane; for the toxins contained in the pus are distributed throughout the body, and set up an enteritis, with its train of evil consequences. Hartmann [1] investigated these conditions in the Berliner Kinderklinik, and found, as Ponfick had, that purulent otitis media and intestinal catarrh react on each other so directly that paracentesis and evacuation of the secretions "may be followed by return of the disturbed digestive function to the normal, and an increase in weight instead of a loss." In one case of acute otitis media, when a second rise in temperature clearly indicated paracentesis, the intestinal condition presented a perfect reflection of the state of the suppurative process. On two other occasions retention of the pus was followed by indigestion and a loss of weight, but both conditions immediately began to improve after paracentesis had been performed. Auto-infection may also result, as Ponfick has shown, from swallowing the pus that has reached the pharynx through the Eustachian tubes.

Hartmann rightly concludes from his observations that in all intestinal diseases of infants accompanied by rise in temperature and loss of weight the ears should be examined to ascertain whether any inflammation is present.

Brieger [2] mentions the rare occurrence of *icterus* in the course of a genuine otitis media, and explains it by the decomposition of blood-corpuscles in hemorrhagic exudations in the tympanic cavity. A slight icteric discoloration of the skin, which may be explained in a similar way, is rarely seen a few days after an operation on the mastoid process.

[1] "Verhandl. der D. otol. Gesellsch.," 1898, p. 87.
[2] "Klin. Beitr. zur Ohrenheilk.," p. 64.

IV. DISEASES OF THE BLOOD.

1. ANEMIA.

The three forms of anemia—simple or symptomatic anemia, chlorosis, and pernicious anemia—will be discussed together, as the symptoms they produce in the organs under discussion are essentially the same.

A constant symptom noted by inspection of the upper air-passages is a marked pallor of the mucous membranes, which may be very intense even in the nose, where anemia does not, as a rule, produce any noticeable alteration. In acute anemia after hemorrhage, and in the anemia of starvation, olfactory hallucinations and exaggerated sensitiveness of the olfactory nerves [1] are observed. These are probably analogous to the rarely mentioned auditory hallucinations,[2] and, though we are unable to explain them, cerebral anemia is no doubt the cause.

The mucous membrane of the pharynx and larynx, especially in chlorotic subjects, is often the seat of hyperesthesia and paresthesiæ, such as dryness and tickling in the throat, exciting cough and hawking. Chlorotic young girls often complain that the voice is weak, is easily tired by talking and even more by singing, and that it becomes hoarse.

The laryngoscopic image often shows nothing but a slight insufficiency of the vocal muscles and of several adductors. It seems probable that this functional aphonia is merely the expression of a weakened state of the muscles due to the anemia, for laryngoscopic examination often shows that the cords move perfectly with the first efforts at phonation; paresis developing only after a number of movements have been made, as the muscles become fatigued very rapidly.

Among aural symptoms in anemic states are tinnitus and vertigo, and, more rarely, difficult hearing, which may go on to total deafness. Opinions are divided on the question

[1] Hoffmann, "Lehrb. der Constitutionskrankh.," pp. 19 and 36.
[2] Haug, "Krankh. des Ohres," p. 176.

of the seat of these disturbances. The results of a functional examination point to disease of the internal ear, but no characteristic signs are elicited. The anemic disturbances of the hearing have been attributed to anemia of the labyrinth, which is assumed to give rise to the symptoms of tinnitus, difficult hearing, and vertigo ; but no satisfactory explanation has been offered of the way in which anemia of the labyrinth could produce such phenomena.

As most cases of grave anemia are, in fact, associated with tinnitus and vertigo, it is quite natural to regard these symptoms as the expression of an anemia of the labyrinth, which has brought on a pathologic condition of irritation in the end-organs of the auditory nerve in the labyrinth. This view of the origin of tinnitus and vertigo finds some support in a case reported by Lermoyez,[1] in which the tinnitus disappeared and hearing improved after food was taken, but the symptoms reappeared in a few hours, with returning inanition. Impaired hearing from anemia may perhaps be interpreted as the expression of a nutritive disturbance in the organ of Corti, which would explain such cases as Abercrombie's, in which the patient was deaf in the sitting posture, but regained his hearing perfectly on lying down ; but there is another possible explanation for this case as well as for Litten's,[2]—where a chlorotic subject suffered with deafness lasting several hours, sometimes, but not always, after a fainting fit,—namely, that the deafness is due to anemia of the deep nucleus of the auditory nerve in general anemia of the brain. These periodic attacks of deafness—which may occur without any permanent lesions of the auditory apparatus, as shown by the fact that the deafness is variable and eventually ends in recovery—are in marked contrast to deafness coming on suddenly after a severe hemorrhage, in which the prognosis is very unfavorable. Such sudden deafness after profuse bleeding at the nose was observed by Urbantschitsch[3] ; it also occurs in greater or less degree after difficult labors attended with great loss of blood. It can not be explained as a result of the sudden change in blood pressure, and must be attributed to a more profound lesion. Some light has been thrown on its mode of origin by the discovery of hemorrhages in the

[1] "Ann. des mal. de l'oreille," 1896, part II, p. 28.
[2] "Bleichsucht," Nothnagel's "Spec. Path. u. Ther.," p. 97.
[3] "Arch. f. Ohr.," XVI, p. 105.

labyrinth by Habermann[1] in a series of autopsies on subjects who had died of simple and pernicious anemia. These hemorrhages appear to be analogous to those found in anemia in the spinal marrow, in the medulla, and in the nerve-trunks.

The theory which seeks to explain that tinnitus aurium in anemia and chlorosis is the noise of the blood stream perceived by the patient himself lacks confirmation, and does not seem probable, as tinnitus aurium is not a constant feature in chlorosis with vascular murmurs. The condition which obtains when the vessels are diseased has been explained elsewhere, and we will only mention here that the variation in the perception of subjective noises in chlorosis has been attributed by Wolf to differences in the conducting power of the bone, depending possibly on imperfect development of the mastoid cells.

Lermoyez suggests the inhalation of amyl nitrite as a diagnostic aid in determining whether tinnitus and vertigo, in a given case, are due to anemia, as its administration is followed by hyperemia and consequent disappearance of the symptoms.

2. LEUKEMIA.

ALTERATIONS IN THE UPPER AIR-PASSAGES IN LEUKEMIA.

Associated with the waxen hue of all the mucous membranes in leukemia there is a peculiar yellowish pallor of the upper air-passages, more conspicuous in the pharyngeal cavity and in the larynx than in the nose, where changes of color are not so marked. As an expression of the hemorrhagic diathesis in leukemia we frequently have epistaxis, which may occur at any stage of the disease without appreciable macroscopic alterations in the nasal mucous membrane, but appears to be most common in the acute form of leukemia, which has lately become better known through the investigations of Ebstein. Microscopically, Suchanneck [2] found lymphoid infiltrations in some of the arterioles of the nasal mucous membrane, and large accumulations of pigment around the vessels. Similar

[1] "Prag. med. Wochen.," 1890, No. 39.
[2] "Zeitschr. f. Ohr.," XX, p. 42.

hemorrhages are found in the external skin and in the mucous membranes, as well as in the pharynx and larynx.

But in addition to these minor changes, during leukemia we find in the pharynx and larynx lymphoid nodules, lymphomatous infiltrations of the mucous membranes with secondary necrosis, and ulceration, making up a clinical picture of genuine leukemic pharyngitis and laryngitis. Virchow's [1] description of the condition has become classic, and well deserves quoting: "Lymphoid nodules appear on the inner surface of the epiglottis, on the aryepiglottic folds, and over the entire surface of the larynx and trachea, sometimes even in the bronchi, presenting usually a small, whitish, moderately raised and rounded swelling of rather soft consistency, frequently situated at the orifices of gland ducts, but also found in other situations." The nodules are usually discrete, but occasionally they coalesce and form a dense uniform infiltration, as observed by Virchow in the upper segment of the larynx. Sometimes they attain to a large size and form tumors. Such tumors, having the consistency of marrow and a glossy surface, are found on the mucous membrane of the pharynx, at the base of the tongue, and on the tonsils.[2] Thus, hypertrophy of the palatal and pharyngeal tonsils is often a valuable sign of leukemic pharyngitis. Virchow remarks that the nodules show no tendency to undergo fatty or cheesy degeneration, and thereby distinguish themselves from miliary tubercles, which they resemble in external appearance. We also have superficial ulcerations (as in the intestine), which, although they also more rarely affect the epiglottis,[3] and have been observed in one instance on the vocal cords in the form of flat ulcers with thickened and slightly reddened edges,[4] show a predilection for the fauces and follicles of the tongue. In some cases the tonsils and fauces take on a dark red, livid color, become greatly swollen, and then undergo necrotic disintegration. This is often associated with a gangrenous form of stomatitis and gingivitis, which strongly suggests grave mercurial intoxi-

[1] "Krankh. Geschwülste," vol. II, pp. 569 and 574.

[2] Recklinghausen, "Virch. Arch.," vol. XXX, p. 370. Mosler, "Virch. Arch.," vol. XLII, p. 445.

[3] Fränkel, "Deutsche med. Wochen.," 1895, p. 679. Kraus, Nothnagel's "Spec. Path. u. Ther.," XVI, I. Th., I. Abth., p. 291.

[4] v. Recklinghausen, "Virch. Arch.," XXX, p. 370.

cation or scurvy (Kraus).[1] It is of comparatively frequent occurrence in acute leukemia, and is probably due to bacterial infection of the mucous membrane which has been deprived of its superficial epithelium by some mechanical trauma and, owing to its impaired nutrition, is unable to offer any resistance to the invasion of pathogenic germs.

Löri [2] and Hoffmann [3] have reported paralyses of the recurrent nerve from pressure or traction of the leukemic tumors on the vagus or recurrent.

The same alterations are found in *pseudoleukemia* as in leukemia, but the literature on the subject is very scanty. Stieda [4] and Kümmel [5] observed diffuse infiltrations, which in the former's case led to a stenosis requiring tracheotomy for its relief, and in the latter's presented a peculiar marrow-like appearance and caused a thickening of the entire mucous membrane, as well as of the aryepiglottic folds and the posterior arc of the entrance to the larynx, and led to a laryngeal stenosis. In a case reported by Kraus [6] the mucous and muscular tissues of the pharyngeal vault and posterior nares were replaced by a hard, whitish mass, slightly raised above the level of the surrounding parts. Necrotic disintegration of the tonsils has occurred in pseudoleukemia, and hemorrhages from the nose, pharynx, and larynx are sometimes observed.

Contrasted with these diffuse pseudoleukemic infiltrations we meet with circumscribed lymphatic tumors on the epiglottis and on the base of the tongue, as observed by Beale [7] and Eppinger [8] in general lymphomatosis.

With symptoms such as these, which in Eppinger's case led to a clinical diagnosis of multiple carcinomatosis, one may well hesitate whether to ascribe the neoplasms to pseudoleukemia or to consider them as idiopathic malignant tumors.

There is another form of morbid growth, known as *lymphosarcoma*, to which it is even more difficult to assign a place among the pseudoleukemias. According to Kun-

[1] Kraus, Nothnagel's "Spec. Path. u. Ther.," XVI, 1.Th., 1. Abth., p. 291.
[2] "Die Veränderungen des Rachens," etc., p. 94.
[3] "Lehrb. der Constitutionskrankh."
[4] "Arch. f. Laryng.," IV, p. 46.
[5] "Verhandl. der D. otol. Gesellsch.," 1896.
[6] *Loc. cit.*, p. 303.
[7] Quoted from Stieda, "Arch. f. Laryng.," vol. IV.
[8] In Klebs' "Handb. der path. Anat.," 7th ed., 1880, p. 209.

drat,[1] "lymphosarcoma is more closely allied to lymphoma, especially of the pseudoleukemic variety, than it is to sarcoma, although it differs from the former by its atypical structure, its mode of growth, and its tendency to invade neighboring tissues." The close relation existing between pseudoleukemia and lymphosarcoma is shown by the tendency of pseudoleukemic lymphomata to change into lymphosarcomata. Kundrat describes them as originating in lymph glands (which consist of follicular and adenoid tissue) in certain regions, following the course of the lymph-channels in their subsequent growth. Lymphosarcomata often originate in the structures of the pharynx; and, according to Störk,[2] the disease frequently begins as a hyperplasia of the pharyngeal tonsil, simulating the picture of adenoid vegetations. The general appearance of the patients, their pallor and cachexia, and the enlargement of the lymphatic elements in the mesentery and retroperitoneal space and of the lymphatic glands generally, which is found at the autopsy, point to leukemia, although the differential diagnosis is indicated by the absence of hepatic and splenic alterations. The tonsils and the follicles of the tongue and of the posterior pharyngeal wall may become enlarged, or an extensive infiltration distributes itself over the posterior and lateral walls of the pharynx, and appears in the larynx either primarily or as an extension from the pharynx. The infiltrated areas usually become the seat of tumors, which differ from similar growths in leukemia in their tendency to cicatrization. In the literature there is no record of hemorrhages.

THE MANIFESTATIONS OF LEUKEMIA IN THE EAR.

It has been known for some time that the ear sometimes becomes diseased in the course of leukemia. Vidal and Isambert found auditory disturbances in three out of thirteen and in four out of forty-one cases, respectively, but in the absence of reliable clinical observations and anatomic studies the nature of the aural disease and its connection with leukemia remained shrouded in mystery. In 1884 Politzer published a paper on the subject, and since then a

[1] "Wien. klin. Wochen.," 1893, Nos. 12 and 13.
[2] Nothnagel's "Spec. Path. u. Ther.," XIII, 2. Th., 1. Abth.; vol. I, p. 204.

few other cases were reported. Finally, Schwabach [1] contributed a decided addition to our knowledge of leukemic disease of the ear by five observations of his own, with anatomic notes, and thereby brought the total number of cases reported up to fifteen.

It has been mentioned that, according to Vidal and Isambert, the proportion of aural complications in leukemia is 10%; Schwabach puts it at 33%, as his five cases of ear disease represent the proportion among fourteen cases of leukemia. F. A. Hoffmann [2] also considers disturbances of the hearing fairly common in leukemia.

The aural disease may appear at any time in the course of the general disease, but is most frequent in chronic cases during the last few weeks before death.

The auditory disturbance is usually profound and points to disease of the internal ear; as a rule, the onset is sudden, with vertigo, tinnitus aurium, and sometimes vomiting, and is immediately, or within a few days or hours, followed by marked reduction in the hearing or even by total deafness. In many cases, including the five out of the fifteen reported by Schwabach, the aural phenomena made their appearance suddenly, simulating the picture of Ménière's symptom-complex.

It has not as yet been determined just how far one is justified in assuming a causal relation between leukemia and these attacks of deafness which do not present any definite clinical type. The assumption that there is a true leukemic form of ear disease is amply justified by the investigations of Schwabach, who found in fourteen of the fifteen cases examined so far anatomic alterations which were undoubtedly dependent on the leukemia.

These changes, which were also observed by F. A. Hoffmann, consist in hemorrhages and in lymphomata situated not only in the *labyrinth*, but also in the *auditory nerve* and its branches. Aggregations of leukocytes or lymphatic infiltrations with extravasations of blood were frequently found in the trunk of the auditory nerve,[3] in the cochlea and vestibule, and in the semicircular canals; sometimes pigmentation was present—a consequence of former hemorrhages. The marrow-spaces in the spongy tissue of

[1] "Zeitschr. f. Ohr.," XXXI, p. 103.
[2] "Lehrb. der Constitutionskrankh.," p. 79.
[3] Alt und Pineles, "Wien. klin. Wochen.," 1896, No. 38.

the mastoid process may be filled to bursting with mononuclear leukocytes, interspersed with hemorrhagic extravasations. It would appear that these masses sometimes organize and are converted into connective or bony tissue.

A unique case is reported by Kast [1] : The labyrinth and auditory nerve were intact, but in the medulla there was an area corresponding to the olivary nucleus, and to the nuclei of the hypoglossus, glossopharyngeal, vagus, auditory, and facial nerves, in which the medullated nerve-fibers were diminished. Yet here bulbar phenomena had not been observed, for the only clinical symptoms were impaired hearing and facial paralysis. Facial paralysis was also present in one of Schwabach's cases.

The complications of the middle ear are less pronounced and less frequent, and the external meatus and tympanic membrane scarcely ever present alterations referable to leukemia. If we disregard the deviations from the normal observed in the otoscopic image,—calcifications or opacities which had nothing to do with the leukemic process,—we find few instances of hemorrhage or injection of the membrane and external auditory meatus. Occasional extravasations of blood, with a variable admixture of red and white blood-corpuscles, have been observed ; more frequently the mucous membrane of the middle ear was thickened, but it was rarely the same extensive leukemic infiltration as that which occurs in the internal ear.

These findings are sufficiently characteristic to remove any doubt that ear disease of leukemic origin is possible. But there is no reason for adopting the opinion of Gradenigo,[2] based on three cases, that an inflammatory process in the ear must be regarded as an essential predisposing factor of ear complications in leukemia.

Examination with the tuning-fork is of the greatest importance in the diagnosis of leukemic ear disease ; there are, it is true, a few cases where the hearing was only slightly impaired, even for whispered sounds, but they are very exceptional compared to those which are characterized by great reduction in the hearing or even total deafness. Without the results of the functional test, which incidentally enables us to determine whether the sound-conducting or the sound-perceiving apparatus is chiefly

[1] "Zeitschr. f. klin. Med.," 1895.
[2] "Arch. f. Ohr.," XXIII, p. 261.

affected, the symptoms of tinnitus and vertigo are of no value in the diagnosis of leukemic disease of the organ of hearing, as both phenomena may be present in this as in other morbid states of the blood-producing organs, especially in anemia, quite independent of any organic lesion in the ear.

The prognosis is unfavorable, though there may be temporary improvement in the hearing.

Our knowledge of auditory disturbances in pseudoleukemia is very limited. Kümmel[1] reports a case which he observed very carefully, and in which the tympanic membrane was dark blue, almost violet in color, the handle of the malleolus being very distinctly seen. At the autopsy an extravasation of blood, mixed with leukocytes, was found in the middle ear. In a case of Hodgkin's disease reported by Brauneck[2] it is said that the hearing, which had always been bad, became worse toward the end, and the diagnosis of disease of the labyrinth or of the central organs was made by an ear specialist.

3. HEMORRHAGIC DIATHESES.

In the hemorrhagic diatheses—*hemophilia*, *purpura*, and *scorbutus*—the same processes are found in the mucous membranes as in the skin. Ecchymoses and hemorrhages may appear in the mucous membranes of the upper air-passages, just as they attack the external auditory meatus, the tympanic membrane, and the middle ear.

These complications are, however, rarely observed, and their diagnosis, when they appear in connection with the primary disease, presents no difficulties, so that nothing would be gained by giving a detailed description, and I shall content myself with presenting a few examples of the individual varieties, culled from the literature.

Epistaxis occupies the first place among spontaneous hemorrhages from mucous membranes in hemophilia; among 236 hemorrhages of various kinds 122, according to one authority,[3] were from the nose. In the same place

[1] "Verhandl. der D. otol. Gesellsch.," 1896.

[2] "Deutsches Arch. f. klin. Med.," vol. XLIV, p. 297.

[3] Quoted from Hoffmann, "Lehrb. der Constitutionskrankh.," p. 121, No. 43.

a quotation is found from Eichhorst, to the effect that the hemorrhage may be preceded by perversions of the senses of taste and smell; one patient could smell, another taste, the approach of his hemorrhage.

I have seen hemorrhages from the larynx in a bleeder, a young woman of twenty-five, in association with periodic subcutaneous and other hemorrhages. The patient expectorated blood, and in the laryngoscopic image the blood could be seen trickling from a point at the posterior extremity of the left false vocal cord and spreading over the adjacent parts, while the entire mucous membrane, including the true vocal cords, showed marked redness. The attacks usually lasted from one to two days, and during the intervals of freedom from hemorrhage the laryngeal image was entirely normal and the source of the hemorrhage could not be recognized.

An excellent example of hemophilic alterations in the ear is furnished by a case of Rohrer's,[1] in which there were hemorrhages in both tympanic membranes, which were dark red in their entire extent. A week later there was another hemorrhage in both membranes, which were deeply injected; on the left side the membrane was dark red, almost black, in color, with the handle of the malleolus sharply defined in white against the dark background—a sign that there was a hemorrhage in the middle ear. Haug [2] reports one case of hemorrhage lasting several hours from rupture of the ear-drum by a blow, and another in which minute punctiform ecchymoses appeared in both membranes after an attack of sneezing. These alterations may, however, occur in anybody, whether he be a bleeder or not, and are not in any sense to be considered characteristic of hemophilia.

In purpura hæmorrhagica the occurrence of epistaxis,[3] ecchymoses, and subcutaneous hemorrhages in the larynx, as well as of ulcerations in the pharynx and larynx, has been reported. Krieg [4] gives a reproduction of hemorrhage on the laryngeal surface of the epiglottis in purpura; Schnitzler,[5] a picture of diffuse hemorrhages from the true and false vocal cords in morbus maculosus Werlhofii.

[1] Reported in "Arch. f. Ohr.," XXXII, p. 59.
[2] "Die Krankh. des Ohres," p. 179.
[3] E. Wagner, "Deutsches Arch. f. klin. Med.," XXXIX, p. 475.
[4] "Atlas," Pl. III, Fig. 7.
[5] "Atlas," Pl. II.

Musser [1] mentions inflammation of the throat simulating diphtheria in purpura. Kaposi [2] saw an ulcer on the epiglottis. E. Wagner [3] described extensive ulcerations in the larynx and pharynx ; and the laryngoscopic examination showed marked turgescence and intense redness of the epiglottis and aryepiglottic folds, and at the autopsy there were found ulcers varying in size from a split pea to a dime, some with a granulating and others with a smooth base, situated on the vocal cords, the aryepiglottic fold, near the free border of the epiglottis, and on the posterior and lateral walls of the pharynx and velum palati. Wagner regards these ulcers as the expression of a process analogous to a cutaneous erythema.

Moos [4] and Haug [5] are the only ones who have described alterations in the ear. The former found a hematotympanum with ecchymoses in the bulging tympanic membrane; the latter, petechiæ in the cochlea and external auditory meatus and on the tympanic membrane.

According to Litten,[6] severe attacks of epistaxis occur in *scorbutus*, which require tampons to control the hemorrhage and may lead to a fatal issue. The hemorrhage is said to be more apt to occur after a slight injury to the nasal mucous membrane or violent blowing of the nose than spontaneously.

Truckenbrodt [7] reports the autopsy of a man who had died of scorbutus ; the patient had not been examined *in vivo*, but had never complained of tinnitus or pain in the ear. An extravasation of blood was found in the dermic layer of the right tympanic membrane ; the mucous membrane of the middle ear was puckered and contained a hemorrhage, and a few petechial hemorrhages were found in the mastoid antrum.

[1] Schmidt's "Jahrb.," CCXL, p. 244.
[2] "Semon's Centralbl. f. Laryng.," II, p. 476.
[3] "Deutsches Arch. f. klin. Med.," vol. XXXIX, p. 467.
[4] Schwartze's "Handb.," I, p. 547.
[5] "Die Krankh. des Ohres," p. 178.
[6] Nothnagel's "Spec. Path. u. Ther.," VIII, I. Th., p. 298.
[7] "Arch. f. Ohr.," XX, p. 265.

V. CHRONIC CONSTITUTIONAL DISEASES.

1. RACHITIS.

It has always been the custom to regard laryngeal spasm as a symptom of rachitis, but in recent years a literary controversy was provoked by the writings of Escherich and Loos,[1] and there is now a movement in favor of treating laryngeal spasm as a symptom of tetany, denying any etiologic relation with rachitis.

An analysis of all reported cases, however, shows beyond a doubt that rachitis exists in the great majority of cases of laryngeal spasm,—three-fourths of all cases according to some authorities, 90% according to others,—and it is preposterous to ascribe this coincidence entirely to accident. Loos himself, although he denies any causal relationship, states that the children affected with spasm of the glottis "as a rule exhibit distinct signs of rachitis."

Laryngospasm, or spasm of the glottis, is an expiratory apnea occurring usually in children under two years of age. The attacks come on suddenly, without ascertainable cause, last from a few seconds to about half a minute, and end abruptly, with a deep whistling or with several rapid, superficial inspirations, after which quiet breathing is restored. The child assumes a rigid attitude, with head thrown back, eyes fixed and staring upward, arms extended, and hands clenched; the face becomes cyanotic and wears a look of extreme fright—in short, we have the terrifying picture of complete asphyxia. But the attack, although it seems very alarming, usually subsides, and only in rare instances terminates fatally.

The whole clinical picture shows that we have more than a simple spasm of the adductors of the vocal cords to deal with, in which the dyspnea is due solely to occlusion of the glottis. In the latter form—which we observe, for instance, after endolaryngeal interference—the integrity

[1] "Deutsches Arch. f. klin. Med.," vol. L.

of the respiratory muscles is retained, as we see by the distinct voluntary inspiratory movements; but in laryngismus stridulus of infants the expiratory muscles and the diaphragm are also involved in the spasm. It is, therefore, not a spasm of the larynx, but, to quote Rehn,[1] a spastic symptom-complex, for which no appropriate name has as yet been discovered. The term "tetanus apnoicus infantum," suggested by Elsässer,[2] has the objection, in these days of controversy on the subject of tetany and rachitis, of appearing to take the etiology for granted; and Oppenheimer's [3] "asthma rhachiticum," while it has the same objection, is also misleading, as the condition it is intended to designate in no way resembles asthma.

Laryngoscopic examination during the attack is out of the question, and the assumption that the glottis is convulsively closed during the attack rests on a purely speculative basis. Schrötter, [4] who is an adept in laryngoscopic technic, says that spasm of the glottis is not a subject for laryngoscopic examination, from which it may be inferred that he never saw a laryngeal image in this affection, and it is therefore the more surprising that Löri,[5] without even alluding to the difficulties attending the examination, and the possibility of failure, gives the following description: "During the attack I always found the rima glottidis tightly closed, but the closure in every case was effected by the true vocal cords alone, without the aid of the false cords. The epiglottis was always depressed, as is constantly the case in very young children, except when they are crying in a very high key, or choking or drawing breath with a whooping sound; but the depression was never complete, so that in most cases I could see the posterior segments of both vocal cords, and in some instances the entire posterior half. I have never seen the epiglottis wedged in between the arytenoid cartilages."

Various views have been advanced on the mode of origin of spasm of the glottis. Some seek the cause in rachitic changes and the rachitic diathesis [6]; others in disturbances

[1] "Berlin. klin. Wochen.," 1896, No. 33.
[2] Quoted by Flesch, "Gerh. Handb. der Kinderkrankh.," p. 289.
[3] "Deutsches Arch. f. klin. Med.," XXI, p. 559.
[4] "Krankh. des Kehlkopfes," 1st ed., p. 386.
[5] "Veränderungen des Rachens, des Kehlkopfes und der Luftröhre," p. 99.
[6] Kassowitz, "Wien. med. Wochen.," 1893, p. 545. Vierordt, Nothnagel's "Spec. Path. u. Ther.," vol. VII, I. Th.

of the digestion [1] more or less closely dependent on rachitis; still others in a nervous predisposition [2]; and some, finally, reject rachitis altogether and attribute the phenomenon to tetany.[3]

Rehn takes a middle view, and attributes the spasm to irritation of the sensory fibers of the vagus by toxins elaborated in the stomach as the result of faulty metabolism. As the origin of this symptom-complex—which, although its etiology is still very obscure, has been termed infantile tetany—has been thought by some authorities to be due to the action of toxins manifesting itself in digestive disturbances, we see in this proposition of Rehn's the possibility of a uniform etiology for that hitherto antagonistic tripod—rachitis-laryngospasm-tetany.

It is admitted by everybody that malnutrition is a predisposing factor, or even an exciting cause, of spasm of the glottis, and it has been found by experience that the most successful treatment of laryngospastic attacks consists in regulating the nutrition.

Since the spasm is not limited to the larynx, but merely forms a part of the general convulsions which play so important a part in rachitis, it can not be regarded as the effect of irritation of a definite portion of the peripheral or central nerve paths presiding over the action of the laryngeal muscles; and until the etiology is better understood, it is idle to suppose a cortical irritation or a lesion in the medulla or in the pneumogastric. There is little foundation either for Kassowitz's theory that spasm of the glottis is due to irritation of the cortical centers (described by Semon-Horsley, Krause, and Unverricht-Preobraschensky) by a hyperemic, inflammatory condition of the rachitic cranial bones, or for that of Oppenheimer, which assumes some irritative action of the jugular vein on the vago-accessorius nucleus due to rachitic alterations at the jugular foramen. The most we can say is that spasm of the glottis in children is the expression of an abnormal excitability of all the respiratory muscles, and that it often occurs, in association with tetanic symptoms (Chvostek, facial nerve phenome-

[1] Flesch, "Spasmus glottidis," in "Gerh. Handb. der. Kinderkrankh.," 1879. Rehn, "Berlin. klin. Wochen.," 1896, No. 33. Hauser, "Berlin. klin. Wochen.," 1896, No. 35.

[2] Flesch, "Spasmus glottidis," in "Gerh. Handb. der Kinderkrankh.," 1879.

[3] Loos, "Deutsches Arch. f. klin. Med.," L, p. 169.

non), in rachitic subjects as the result of digestive disturbances.

Of the relations between *rachitis* and *aural disease* nothing positive is known. The attempt to establish a connection between the former and purulent or catarrhal disease of the middle ear has been made, but there is not a shadow of proof to justify it. Such superficial statements as those made by Ertelberg, and faithfully repeated by Haug, are of no value whatever; for the mere fact that among 250 rachitic children there were 25 cases of middle-ear disease and only 27 absolutely normal tympanic membranes,[1] especially when the histories were not altogether negative in the matter of previous infectious diseases, or that "among 180 rachitic children purulent otitis media was found 16 times, otitis externa twice, eczema 9 times, otitis media catarrhalis 19 times, and catarrh of the tubes even more frequently" (Haug[2]), is not in the least significant, as the same conditions are found, even without rachitis, in the children who make up ordinary polyclinic material.

It is quite possible that the general nutritive disturbances and frequent attacks of bronchial catarrh which characterize the course of rachitic disease tend to produce a favorable soil for the development of aural complications, but we are very far from possessing any scientific proof that such is actually the case.

2. ACROMEGALY.

In acromegaly [3] a hyperplasia of the submucous and intermuscular connective tissue takes place, which produces certain alterations in the bones and cartilages. These changes affect more or less the nose, pharynx, and larynx, and to some extent the ears.

Besides the external changes in the nose, which consist in an abnormal increase of the cartilaginous and bony portions, there is hypertrophy of the nasal mucous membrane. The tongue becomes enormously enlarged, and hyperplasia of the submucous tissue in the soft palate takes place. The

[1] "Jahrb. f. Kinderheilk.," XXVII, p. 96.

[2] "Die Krankh. des Ohres," etc., p. 173.

[3] Sternberg, "Zeitschr. f. klin. Med.," XXVII, p. 86. Sternberg, Nothnagel's "Spec. Path. u. Ther.," VII, 2. Th.

larynx is increased in size, as we can determine by external palpation, and the voice is unusually deep and rough and is stronger than normal. As there is no visible alteration in the laryngoscopic image, these phenomena are probably due to the general enlargement of the larynx, to hypertrophy of the mucous membrane, and in part to increased resonance of the voice from the greater volume of air in the chambers of the upper air-passages (Marie).

Sternberg [1] describes diminutions in the caliber of the *external auditory meatus* from *exostoses*, quotes similar observations by Osborne, and adds that the bony portion of the meatus was unusually deep on account of hyperostosis of the bony parts of the skull. As these alterations have been found in acromegalic skulls in several instances, Sternberg believes himself justified in including them among the constant objective symptoms of the disease.

3. DIABETES MELLITUS.

In diabetes the dryness of the oral mucous membrane of which the patients complain finds its counterpart in a dry pharyngitis with redness of the mucous membrane, which, like chronic pharyngitis, is regarded by M. Schmidt[2] as an early symptom of the disease. Löri [3] claims that the same condition of dryness and atrophy may be found in the larynx. In this connection it is worth mentioning that *aphasia* has occasionally been noted in association with diabetic hemiplegia[4]; F. A. Hoffmann [5] includes paralysis of the vocal cords among diabetic palsies, but I have not been able to find any case of it in the literature.

Furunculosis and pruritus occur in the auditory meatus as they do in the external skin (Wolf,[6] Haug[7]). If the former recurs frequently, it is said to be a sign of diabetes; but the diagnostic value of this statement is open to question when we contrast the frequency of furunculosis in the

[1] "Zeitschr. f. klin. Med.," XXVII, p. 139.
[2] "Krankh. d. ob. Luftwege," 2d ed., p. 226.
[3] "Veränderungen des Rachens und Kehlkopfes," p. 97.
[4] Charcot, "Arch. de neurolog.," May, 1890. Blanchet, "Gaz. des hôpit.," 1885.
[5] "Constitutionskrankheiten," p. 316.
[6] "Arch. f. Ohr.," p. 166. [7] "Die Krankh. des Ohres," etc.

ear with the rarity of furunculosis of the auditory meatus in diabetes. I have never observed it myself, nor seen it mentioned in any good case history. Blau[1] reports a case in which attacks of furunculosis kept recurring for years without his ever being able to demonstrate any signs of diabetes. Neuralgia of the mastoid process is mentioned among the complications of diabetes by Brieger[2]; it is, however, of secondary importance.

On the other hand, the middle ear and mastoid cells are sometimes attacked by a disease which presents certain characteristic appearances, and justifies the assumption that it is more or less closely related to diabetes. Toynbee describes a case of suppuration of the mastoid process in which extensive carious destruction of the structure was found after death, without, however, referring it to the diabetes which was present at the same time. How recent our knowledge of diabetic ear disease really is appears from the remarks of Senator[3] and Blau, published in 1876 and 1883 respectively, to the effect that loss of hearing and implication of the organ of hearing generally must be very rare in diabetes, to judge from the lack of reported experiences. Naunyn,[4] on the contrary, in his recently published work on diabetes devotes an entire section to diabetic ear diseases, showing how much our knowledge of such complications has advanced in the short space of twelve years. To Kirchner,[5] and even more to Kuhn[6] and Körner,[7] we owe the first discussions on the subject, and to-day we have a goodly number of instructive observations at our disposal which afford certain definite conclusions. The disease is characterized by the sudden onset of violent pain, localized in the ear or, more frequently, in the mastoid. The patients are usually quite unable to give any cause for the pain. In some cases the affected ear was quite healthy before the attack; in others, there is a history of antecedent purulent otitis media. After a longer or shorter interval of pain, usually on the third to the fifth day, perforation takes place spontaneously and pus is discharged. The secretion contains nothing that may not be present in any acute sup-

[1] "Arch. f. Ohr.," XIX, p. 208.
[2] "Klin. Beitr. f. Ohr.," p. 115.
[3] In Ziemssen's "Handbuch."
[4] Naunyn, Nothnagel's "Spec. Path. u. Ther.," vol. VII, 6. Th.
[5] "Mon. f. Ohr.," 1884, p. 221.
[6] "Arch. f. Ohr.," XXIX.
[7] "Arch. f. Ohr.," XXIX.

puration of the middle ear. It may be a mixture of blood and serum, seropurulent, or, in a long-standing case, mucopurulent. Raynaud's[1] case began as a copious hemorrhage from the auditory meatus, which was followed by such an abundant flow of serosanguineous, and later serous, secretion, "as is ordinarily seen only in the discharge of cerebro-spinal fluid after trephining," and finally went on to the purulent stage.

In a remarkably short time the morbid process in diabetic otitis spreads to the bones. The rapidity with which the disease is followed by carious disintegration of the mastoid cells is commented upon by Toynbee and, after him, by many other observers; it is even greater, according to Kuhn, than in the most malignant cases of diphtheria. Within the short space of two or three days the interior of the mastoid process in many cases is converted into a large cavity, filled with pus and granulations mixed with sequestra of bone, and in a few weeks the transverse sinus and dura mater of the posterior fossa of the skull are laid bare. Raynaud found, when his case came to the autopsy, the mastoid cells filled with a reddish fluid mixed with inspissated pus, while the mucous membrane was soft and red; in Kuhn's case the bony parts that had escaped destruction were inflamed and so soft that they could be molded and cut like wax. I myself operated on two cases in which the spongy tissue was much discolored and scantily streaked with pus; the bone was very anemic and brittle from necrosis, suggesting the appearance of a preparation which has been in alcohol for a long time. In several places there were large sequestra, which could be easily removed from the surrounding tissue.

Are these clinical pictures such as to justify the assumption of a diabetic form of middle-ear disease, since their only deviation from an ordinary case of purulent otitis media lies in the rapidity of the course and the early implication of the bone? We can not deny that this is an important element, in spite of Brieger's[2] opinion that the intensity of the process is not sufficient warrant for assuming the existence of a special form of disease. Haug[3] tested the aural secretion for sugar, and found it "at least qualitatively" positive (by what methods?); Raynaud, on

[1] "Ann. des mal. de l'oreille," 1881, p. 63.
[2] "Klin. Beitr. zur Ohrenheilk.," p. 112.
[3] *Loc. cit.*, p. 166.

the other hand, found albumin, but no sugar, in the serous secretion. As the most various secretions and excretions of the body have been found to contain sugar in diabetes, Haug's positive results can not weigh very heavily, while Raynaud's negative result is interesting from the fact that an examination of the fluid taken from the edematous scrotum in the same case showed 0.7% sugar.

The point at which perforation of the tympanic membrane occurs varies, and is of no value for diagnosis, as it occurs indifferently in the anterior or posterior half of the membrane (Raynaud).

The course of the suppurative process is characterized, as has been stated, by rapidity of extension to the bone. Arguing from the extensive and rapid destruction of the mastoid processes, with comparatively mild disease of the middle ear, Kuhn and Körner have advanced the opinion that the process in diabetic ear disease begins as a *primary osteitis* of the mastoid, and extends secondarily to the tympanum, thus bringing about perforation.

It is quite natural that the original opponents of the doctrine of a primary mastoid osteitis should oppose such an assumption, but they were reinforced by others (Davidson[1]), who based their objections on a review of the literature.

In favor of Kuhn's theory we have the clinical features and course of the disease, especially the circumstance (insisted on by Körner) that the changes found in the middle ear bear no proportion to the intense degree of destruction in the mastoid process, and the flow of pus subsides as soon as the diseased bone is opened, as I have myself observed in one of the patients I operated on. Another argument in favor of Körner's view is found in the necrotic, gangrenous appearance of the bone, which I have mentioned, and which was equally marked in both my cases; a dry, gangrenous appearance of the tissues being a well-recognized feature of the diabetic diathesis.

On the other hand, it may be urged against the foregoing theory that the resisting power of the tissues to bacterial invasion is diminished by the presence of sugar, which affords a favorable soil for the growth of pathogenic micro-organisms, so that an accidental infection of the mid-

[1] "Berlin. klin. Wochen.," 1894, No. 51.

dle ear finds the most favorable conditions for the spread of the disease. The comparative benignity of the middle-ear affection can be explained by the drainage facilities through the perforated membrane, which are wanting in the mastoid cells, where the carious process accordingly continues its work of destruction. It should also be said, in justice to the opponents of a primary osteitis, that there are cases in which the bone disease appeared late in the course of a chronic purulent otitis media, just as there are others in which an acute suppuration terminated favorably without involving the mastoid cells.

In this connection a case of Naunyn's [1] is peculiarly interesting. In a severe case of diabetes a violent otitis media developed on the fourth day; the patient, a boy of eight, complained of severe headache, and there were marked cerebral symptoms, with vomiting, great hebetude, and "large respiration, as in diabetic coma." Paracentesis was performed on the fifth day and a large quantity of pus was evacuated; recovery followed in a few days. I once saw a similar case in a boy of fourteen, with grave diabetes, who experienced pain in the ear and a slight otorrhea two days before the occurrence of diabetic coma. On the following day, while the coma continued, the flow subsided, and the ear-drums, which were perforated and showed the scars of former lesions, were seen to be slightly swollen and of a uniform bluish-red color, which soon disappeared. Six months later, the same ear was attacked by acute middle-ear inflammation, necessitating paracentesis; after the discharge had lasted about a week the patient again recovered.

In reviewing the facts before us, it appears that there are unquestionably cases of simple diabetic otitis which prove the existence of a diabetic disease localized in the middle ear; but it is equally certain that there are many cases, reported by Kuhn, Körner, and others, which as emphatically justify the assumption of a primary osteitis, especially since we possess the description of a case of diabetic osteitis and multiple periosteitis elsewhere in the body, which confirms the possibility of such primary bone disease in diabetes.

However that may be, whether we have to deal with a

[1] "Diabetes" in Nothnagel's "Spec. Path. u. Ther.," p. 287.

primary osteitis or a primary otitis media, the occurrence of suppuration from the ear in diabetes constitutes a grave complication, which must be combated from the outset with all the means at our command. There was a time when operative treatment of diabetic otitis media was thought to be contraindicated, because a few deaths had been reported. If the wound is properly treated, this fatal result must be charged to postoperative diabetic coma (two out of four cases by Buck[1]), and not, so far as I can see, to the operation itself[2] (one case reported by Sheppard died of intercurrent erysipelas and purulent meningitis[3]). As it is well known that the morbid process in the bone spreads very rapidly in diabetes, without giving rise to any pronounced subjective symptoms, trephining of the mastoid process is indicated whenever the ominous sinking of the posterior wall of the meatus has been present for some time, or deep abscesses have made their appearance in the mastoid process itself. A *high* sugar percentage is, however, an absolute contraindication, as it enhances the danger of postoperative diabetic coma; this is probably the direct result of chloroform narcosis, which is followed by a rise in the percentage of sugar, as observed in Körner's cases and in my own that terminated favorably (from 0.2 to 1.85% in my cases). Since, therefore, the danger lurks in the anesthesia as well as in the operation itself, one should never operate without first reducing the sugar as much as possible by a long course of dieting. Recent experience teaches that in this way we also diminish the danger of sepsis, which, according to Schwartze,[4] "renders the prognosis as to life a doubtful one, even in mild grades of diabetes, because there is danger of an unfavorable postoperative course, ending in sepsis." At all events, it is not great enough to forbid operative interference, any more than the imaginary danger[5] of uncontrollable hemorrhage, which appears to be founded on a case of Moos,[6] in which "the operation was interrupted by an uncontrollable hemorrhage, lasting three-quarters of an hour"—its origin is not stated, and who is to say that it was due to the diabetes?

[1] "Arch. f. Ohr.," XL, p. 138.

[2] I recently saw a death during coma on the fourth day after the operation; at the autopsy a large abscess was found in the deep muscles of the neck.

[3] "Zeitschr. f. Ohr.," XXIX, p. 268.

[4] "Handb.," II, p. 841. [5] Haug, "Krankh. des Ohres," p. 167.

[6] "Deutsche med. Wochen.," 1888, No. 44.

4. GOUT.

The most familiar examples of gouty alterations are the catarrhal phenomena in the pharynx and larynx. They occur most frequently in the form of angina uratica, with dark-red discoloration of the mucous membrane of the uvula, soft palate, the two pillars of the fauces, and the tonsils. Sometimes an acute edema is superadded, as has been observed by Vaton,[1] M. Mackenzie,[2] and Danziger.[3] Solis-Cohen[4] insists on the frequency of pains and abnormal sensations in circumscribed areas of the mucous membrane which appeared to be perfectly healthy, and in which he found only dilated vessels or a dark-red discoloration. Acute attacks of angina uratica always make their appearance two or three days before a typical outbreak of gout, and subside as soon as the gouty joint-affection has declared itself. There is also, as a rule, chronic pharyngeal catarrh, associated sometimes with tophi (Litten[5]).

Gouty disease of the larynx is rarely observed. It manifests itself in a great variety of forms, the inflammatory redness and swelling being often attended with the deposition of urates in the joints and cartilages. The mucous membrane of the vocal cords is involved, as well as that of the rest of the larynx, and not infrequently there are circumscribed swellings in special portions of the larynx. Thus, in a gouty patient I have seen an infiltration of the right ventricular band persist for many years following a laryngitis which had come on after an acute attack of gout. M. Mackenzie[6] observed a gouty inflammation of the left false vocal cord, with granulations, which had been diagnosed as cancer. Virchow,[7] Litten, Morell, and Mackenzie saw gouty deposits: in one case a white body as large as the head of a pin, at the posterior extremity of the right vocal cord; at other times, as infiltrations in the cords and articulations of the larynx. In Mackenzie's case it was the crico-arytenoid articulation that was affected, and the resulting imperfect approximation of the vocal cords gave rise to aphonia. Litten found postmortem marked infiltra-

[1] "Semon's Centralbl.," VIII, p. 85.
[2] "Journ. of Laryngol.," 1889, p. 313.
[3] "Mon. f. Ohr.," 1895, p. 14.
[4] "Semon's Centralbl.," XI, p. 318.
[5] "Virch. Arch.," 66.
[6] *Loc. cit.*
[7] "Virch. Arch.," 44, p. 137.

tion of the same joints and their ligaments (the clinical appearance of the larynx is not given). The gouty process in the cartilages not infrequently goes on to ossification.

Of the gouty alterations in the organ of hearing those which affect the concha have been known a long time, and every physician is familiar with them. In nearly all of Garrod's [1] case histories we find mention of small gouty nodules in the concha, sometimes on the posterior surface, more commonly on the helix and fossa navicularis. The cartilage is said to be the seat of a peculiar induration and of the formation of small softening foci. In some cases there is inflammation of the external auditory meatus (pruritus). The statement that exostoses in the external meatus are due to gout (Kirchner) has never been proved. Judging from the frequency of complaints from arthritic patients to the effect that they suffer from difficulty in hearing, especially progressive loss of hearing and tinnitus, we must infer that other lesions occur in the organ of hearing. We are not inclined to accept angina as the explanation of the loss of hearing in gouty subjects, as suggested by Haug; for there really is not any form of aural complication that might not occasionally be referred to a hypertrophic pharyngeal catarrh. Ebstein's arguments in his treatise on "Aural Vertigo" seem to us more plausible.[2]

The clinical picture of gouty ear disease, which, as has been said, has for its principal features a progressive diminution of the hearing, with tinnitus and vertigo, may be explained in as many different ways as there have been causes assigned for gout itself. It is still a question whether the gouty process is in the middle or in the internal ear; we can not say positively that the chalky deposits seen during life on the tympanic membranes of gouty subjects consist of urates, for the manner in which the morbid process affects the organ of hearing is very imperfectly understood. A specific gouty affection of the organ of hearing may be situated in the tympanic membrane, where the resulting functional disturbance would probably be slight, or in the chain of ossicles in the form of arthritic disease. Unfortunately, we are without anatomic experience on this point, and even the clinical stock of observations at our command is very limited. A case history, to have any statistical

[1] Deutsche Uebersetzung von Eisenmann, p. 101.
[2] "Arch. f. klin. Med.," 58, p. 1.

value in showing a connection between gout and diseases of the middle ear, should contain not only the results of an accurate functional examination, but also some information in regard to the movability of the chain of ossicles.

Brieger [1] reports a case in which the usual prodromata of an attack of gout were followed by an acute otitis media, with marked bulging and swelling of the tympanic membrane, and interprets it as an arthritic process in the articulation, between the malleus and incus. According to Agnano,[2] persons with the gouty diathesis usually develop deafness between the ages of fifteen and twenty.

Still more uncertain are we whether the labyrinth is ever attacked by the gouty process. Since the imaginary hemorrhages which are sometimes supposed to form the basis of the phenomena in the labyrinth, mentioned previously under the name of Ménière's symptom-complex, must be rejected as being without anatomic foundation, the most natural explanation of these symptoms is suggested by the vascular changes which are a constant feature of gout, and we are therefore inclined to seek the cause of these aural phenomena in a primary arteriosclerosis. This view appears to be supported not only by the observations of Ebstein, but also by de Lacharrière's statement that "aural phenomena are most common in persons who, besides being subject to attacks of genuine articular gout, show their inherited gouty tendencies in attacks of gastralgia, dyspepsia, migraine, and neuralgia." Ebstein is right, no doubt, when he says that it must, for the present, remain an open question whether the ear disease in gouty subjects is to be referred to the primary disease, to obesity, or to cardiac changes the result of overindulgence in alcoholic beverages.

ICTUS LARYNGIS OCCURRING IN THE COURSE OF OBESITY, GOUT, AND DIABETES.

That there is a certain relationship between the three constitutional anomalies, obesity, gout, and diabetes, appears from the way in which they manifest themselves in individual members of a gouty family—now under one form, now under another. They produce chronic catarrhal

[1] "Klin. Beitr. zur Ohrenheilk.," p. 77.
[2] "Rev. hebd. de lar.," 1896, p. 703.

changes in the mucous membranes of the upper air-passages, and a peculiar form of neurosis in the larynx, which has been called "ictus laryngis." Their relation to aural vertigo, tinnitus, and progressive chronic loss of hearing has been sufficiently discussed under the head of gout, where reference was made to Ebstein's treatise on the subject.

We shall, however, give a short description of what is known as "laryngeal vertigo," a condition which more frequently comes under the observation of the general practitioner than that of the laryngologist.

By ictus laryngis is meant a sudden attack of syncope of short duration, preceded usually by a slight paroxysm of coughing. It was first described by Charcot in 1876, then by two French writers, Garel and Collet, and by the Italian, Massei, while in Germany up to the present time only a very few observations have appeared (for instance, Schadewaldt's). Charcot proposed the term *vertige laryngé*, and it is still found in many text-books on laryngology, although vertigo itself is one of the rarest features in the symptom-complex; Kurz's suggestion of lipothymia laryngea (laryngeal syncope)[1] has not met with a very favorable reception. The term laryngeal crisis, which has also been suggested, would only cause a confusion of ideas, because it is applied to an entirely different symptom-complex, which, as we shall see, is peculiar to tabes dorsalis.

The attack occurs without warning in the midst of perfect health; it may come on while the subject is working, sitting, standing, walking, or even lying down. Quite frequently the attack comes on after a meal; sometimes the patient is awakened at night by a slight cough, sits up in bed, and has an attack. The description usually given is that the patient feels a tickling sensation in the throat, has a slight attack of coughing, and loses consciousness for a few seconds; the breathing stops and the face becomes cyanotic. If the subject is standing at the time, he falls to the ground; if he is sitting, the head falls forward on the chest. In a few instances the attack was attended with twitching in the muscles of the upper extremity or of the face, but never with biting of the tongue. The duration is very short,—usually a few seconds; the patient does not

[1] "Deutsche med. Wochen.," 1893.

feel unwell after it is over, and goes on with whatever he is doing at the time as if nothing had happened. When questioned, he says he has had an attack of coughing, but does not complain of any other symptom.

The cases reported nearly all refer to men in the fifth decade of life. The predisposing causes usually given are chronic catarrh of the upper air-passages, chronic pharyngitis and laryngitis, occasionally chronic catarrh of the lungs. Schadewaldt emphasizes chronic alcoholism as a predisposing factor, while Garel and Collet attach great importance to constitutional diseases, as gout, obesity, and diabetes. Cardiac changes play an important rôle : Schadewaldt found the heart hypertrophied (cor adiposum) in five of his seven cases. The clinical picture aroused the suspicion in the minds of the observers that they had to deal with an epileptic attack, but subsequent experience has failed to establish any connection whatever with epilepsy. From the fact that an attack can be brought on by introducing a sound into the larynx, and controlled by cocainizing the mucous membrane, it was argued that it must be a kind of reflex neurosis, but the descriptions offered for the reflex arc rest on a purely hypothetic basis. It seems to be proved by the fact that the attack begins with a tickling and burning sensation in the throat, that it is due to irritation of the superior laryngeal nerve. This being the case, it is supposed that the vasomotor center in the medulla is stimulated through the depressomotor fibers of the vagus, and a fall in the blood pressure takes place ; at the same time the irritation is communicated to the cardiac inhibitory center, so that the action of the heart is diminished. These two factors cooperating to produce anemia of the brain, furnish an explanation of the loss of consciousness, which is characteristic of the attack.

Spastic phenomena are altogether wanting, although some observers attempt to explain the attacks as laryngeal spasm, and it is doubtful whether we are, after all, justified in regarding ictus laryngis as a local neurosis of the larynx. The circulatory system unquestionably plays an important part in the etiology, for many of the cases were complicated with heart disease, and a marked predisposition to the attacks was observed in plethoric persons and in those addicted to good living and alcoholic abuse. The frequent occurrence of the attacks during the digestive

pause immediately following a meal also points to the circulatory system. Schadewaldt reports a case which ended fatally; the patient had had an attack of ictus laryngis on the previous day, after supper, but felt so well on the day of his death that he took his customary horseback ride. In the afternoon, however, while engaged in conversation with a companion, he had another slight attack of coughing, lost consciousness, fell to the ground, and died instantly, without exhibiting any other symptoms. No autopsy is given, but the history of cardiac hypertrophy in a robust, alcoholic individual, fifty-nine years old, justifies the diagnosis of death from heart failure.

VI. ACUTE INFECTIOUS DISEASES.

1. MEASLES.

Catarrhal disease of the mucous membranes in the upper air-passages constitutes an integral part of the clinical picture in measles. It takes the form of an exanthema, which always precedes the skin eruption, and is absent, according to Monti,[1] only in children who are very anemic or weakened by previous disease.

Even during the prodromal stage of measles there is a dark-red discoloration of the pharynx and palate; it is irregularly distributed, and is most marked on the lateral and posterior pharyngeal walls and on the pillars of the fauces. The discoloration is also seen on the mucous membranes of the cheeks and lips, where it constitutes Koplick's [2] sign. The redness is accompanied by a feeling of dryness in the throat; on the following day the mucous membrane appears moist and the true exanthema begins to break out. This exanthematous eruption is most marked on the pillars, where it takes the form of small isolated or confluent macules or papules of varying size, elevated above the level of the mucous membrane (Monti). The skin eruption appears usually from twelve to twenty-four hours later, and with its appearance the patches begin to subside. In addition to the redness and swelling, Tobeitz [3] observed a superficial slough, resembling that produced by a mild caustic, which he interprets as an epithelial necrosis. Similar appearances are seen in the larynx; they also accompany other catarrhal diseases, particularly influenza.

The mucous membrane of the larynx presents a bright-red color, in irregular patches, interspersed with fine granular nodules (Gerhardt). This variety of laryngitis usually appears two or three days after the exanthematous erup-

[1] "Jahrb. f. Kinderheilk.," VI, p. 22.
[2] "Deutsche med. Wochen.," 1898.
[3] "Arch. f. Kinderheilk.," VIII, p. 326.

tion, seldom later, and gives rise to hoarseness and cough of a croupy character. The patches of epithelial necrosis mentioned by Tobeitz take the form of erosions and shallow ulcerations on the posterior pharyngeal wall, and are supposed by Gerhardt to be due to mechanical injury of the already loosened mucous membrane by the act of coughing.

Croupous laryngitis is a rare occurrence in measles. Tobeitz saw evidences of very mild forms at autopsies, not severe enough to cause stenosis, rather a shallow croupous deposit; the mucous membrane in these cases was of a bright-red hue, but not much swollen, and the surface was deprived of its epithelium and in places necrotic. Complications of measles with diphtheria and true diphtheric laryngitis are not unknown.

Thanks to trustworthy anatomic investigations, our knowledge of *ear diseases* in the course of measles is more complete than is the case in the other infectious diseases. To Tobeitz, Rudolf, Bezold, and Habermann we are indebted for investigations on the cadavers of children which give us uniform results concerning the nature and mode of spread of aural complications in measles. One valuable feature of these investigations—especially of Bezold's, who examined a large number of cadavers—is the fact that particular attention was paid to the organs of hearing in those cases which during life had presented few, if any, symptoms of disease, so that an opportunity was afforded of studying the earliest stages of the alterations.

In 16 cases examined by Rudolf (and tabulated under Bezold's direction), 17 by Bezold [1] himself, 17 others by Tobeitz,[2] 6 by Siebenmann,[3] and 7 by Habermann,[4] with only two exceptions there were found signs of an *acute otitis media*, which must be regarded as a special localization of the disease. It was found to persist for some time after the appearance of the eruption, for Bezold's cases belong to the period from the third to the thirty-third day of the disease.

According to Bezold's description of these early appearances in disease of the middle ear—and they can frequently be demonstrated in the first three days after the appearance

[1] "Zeitschr. f. Ohr.," XXVIII, p. 209.
[2] "Arch. f. Kinderheilk.," III, 341.
[3] Quoted from Bezold, "Zeitschr. f. Ohr.," vol. XXVIII, p. 249.
[4] "Schwartze's Handb.," vol. I, p. 261.

of the eruption—there is a diffuse injection and turgescence of the mucous membrane, and the tympanic cavity contains more or less fluid. It is an important point that the disease also extends to the lining of the mastoid antrum and cells.

The secretion in the tympanic cavity was never of the purely serous type found in simple occlusion of the tubes, but was mucopurulent or seropurulent or consisted of pure pus. The injection of the blood-vessels was irregularly distributed over the mucous membrane in the form of patches and minute, punctiform extravasations. Occasionally, a fibrinous exudate ("pseudomembrane") was seen. The swelling was less marked than is usual in middle-ear suppurations. Bezold never found the mucous membrane destroyed so as to expose the bone. The tympanic membrane in all the cases described showed a marked resistance to the attacks of the disease, being thickened, but otherwise intact, even in those cases which came to the autopsy as late as the thirty-third day after the appearance of the eruption. We could not expect, therefore, to have any appreciable changes in the otoscopic image at this stage of the disease, and as it does not give rise to any marked subjective symptoms, it is probable that such low grades of inflammation pass off without being observed clinically. The prognosis is good; after the inflammation subsides and the exudate is absorbed the parts are completely restored to their normal condition.

It is not to be inferred, however, that all aural complication in measles run this benign course. We know from practical experience that *acute purulent otitis media with perforation* is a very common sequel of measles, and, if neglected and allowed to become chronic, it may lead to any of the consequences—such as caries of the bone, exuberant granulations, and cholesteatomata—which we are accustomed to see after any suppurative process in the middle ear. To show how wide-spread is the belief among the laity that measles may be followed by disease of the ear, it may be mentioned that in about 3% [1] of all cases of aural disease measles is given as the original cause by the patient or his friends, and that 5.1% of all cases of purulent otitis media are attributed to this disease. Again, that

[1] From Blau and Bürkner.

the otitis of measles is not quite so benign as might be supposed from the slight attention it has received even in medical circles,—there being a general impression that it requires no special treatment,—is shown by the fact that measles is charged with 4% of all cases of acquired deaf-mutism. As has been previously indicated, the otitis that accompanies measles is not especially malignant, and runs much the same course as any other acute or chronic otitis media. Blau succeeded in curing 28 cases of acute purulent otitis following measles without the hearing being impaired. Bone disease with abscess formation is not more common after measles than in ordinary otitis media.

Otitis usually makes its appearance during the stage of desquamation between the second and third week; two cases have been reported in which it appeared before the eruption.

The course of the disease presents nothing characteristic. Blau [1] reports a case of diphtheric disease of the external auditory meatus, without involvement of the middle ear, which appeared five days before diphtheria of the pharynx following measles. Haug [2] describes a primary caries of the mastoid process, with secondary suppuration of the middle ear, which developed during the stage of desquamation. We do not attach much importance to Moos's [3] observations that disease of the internal ear with sudden deafness and vertigo may follow an attack of measles, as they lack the confirmation of other observers.

A review of our knowledge concerning the nature and course of the otitis of measles justifies the following conclusions: It appears, from the results of clinical and anatomic investigations, that there are two varieties of otitis in measles, the second of which represents a complication of the first. The otitis media described by Bezold and others represents a true measle eruption affecting the mucous membranes, while the suppurative process with perforation of the tympanic membrane must be regarded, after Bezold, as the result of a mixed infection which finds a favorable soil in the mucous membrane weakened in its resisting power by the primary disease.

Another view, which is advocated by Wagenhäuser [4] and

[1] "Berlin. klin. Wochen.," vol. XXXIII, 1884.
[2] "Arch. f. Ohr.," XXXII, p. 183. [3] "Zeitschr. f. Ohr.," XVIII.
[4] Quoted by Habermann, "Schwartze's Handb.," I, p. 761.

others, regards the otitis of measles as a simple inflammation derived from the postnasal space through the Eustachian tubes; but in the light of recent investigations on cadavers, this view seems to us to lack general application, although it may hold in isolated cases. The early development of the acute inflammation, coincident with the appearance of the eruption, confirms the hypothesis that we have to deal with a true measle eruption precisely analogous to that on the mucous membrane of the respiratory tract, and worthy of a place in the general symptom-complex in measles. We know from the investigations of Bezold that the catarrhal process in the middle ear runs a very chronic course, and that the mucous membrane shows little tendency to regeneration and granulation; hence, its susceptibility to secondary infection, even several weeks after the measles has run its course, is quite readily understood.

2. SCARLATINA.

Among the complications of scarlet fever in the upper air-passages we distinguish catarrhal angina and a form of diphtheria.

The catarrh of scarlet fever is distinguished from that which occurs in measles by being restricted in the main to the pharynx, faucial pillars, and tonsils, while the nose and larynx usually escape, or, at any rate, become involved much later. It manifests itself as a deep-red or violaceous discoloration, at first uniform, and after a few days distributed in patches; the mucous membrane is dry and very much swollen, causing a feeling of dryness and tickling in the throat and a desire to swallow at frequent intervals. The onset and course of the angina do not appear to follow any definite rule; in most cases it appears before the eruption and lasts several days.

In some cases of malignant scarlatina without eruption, which terminate fatally very soon after the onset of the disease, with grave constitutional symptoms, this dark-red discoloration of the pharyngeal structures may form the only symptom, and its relation to scarlet fever can be determined only by the existence of an epidemic or by the subsequent outbreak of the disease in other members of the family.

The regularity with which this catarrh of the mucous

membrane appears at the very outset of the infectious disease, and its localization in the region of the pharyngeal ring, so abundantly supplied with lymphatic elements, justify the assumption that the virus of the disease, the nature of which is not known, gains entrance to the system at this point, and that the angina of scarlet fever represents the earliest reaction of the organism to the scarlatinal poison.

In uncomplicated cases these catarrhal symptoms subside in a few days, but in a large proportion of cases a streptococcal infection of the diseased mucous membranes is superadded to the scarlatinal poison and gives rise to a group of morbid phenomena which are designated by the general term "diphtheroid scarlatina." It is a necrotic inflammation of the mucous membrane, presenting the anatomic picture of diphtheria, but having etiologically nothing in common with genuine diphtheria, from which it is distinguished by the absence of Löffler's bacilli and by certain clinical differences in the mode of spread and the development of sequels.

Before Heubner's publications appeared to throw some light on the question, the greatest confusion prevailed in the diagnosis and description of diphtheroid scarlatina, the shadow of which overhangs even the most recent rhinootologic literature and materially detracts from the value of reported observations.

Heubner [1] divides diphtheroid scarlatina into three forms, according to the clinical course,—a mild form, a subacute form, and an epidemic form,—which together represent various grades of virulence, both in respect to the extent of mucous membrane involved and to the manner in which the neighboring glands react to the poison. The first form is characterized by the deposition on the first to the third day of small superficial exudates on the surface of the inflamed tonsils; these soon run together and form a delicate membrane, which can be removed with a pair of forceps without causing hemorrhage. After persisting a few days the membrane is replaced by shallow ulcers which rapidly heal, while the swelling of the submaxillary glands subsides.

Heubner observed this favorable course in about one-

[1] "Volkmann's Vortr.," No. 322 (1888); and Hirschfeld, "Jahrb. f. Kinderheilk.," vol. XLIV, p. 237.

fourth of all cases of scarlatinoid diphtheria. In almost all cases the mild form is followed by the so-called subacute (*"lentescent"*) form of scarlatinoid diphtheria (Hirschfeld observed it in 53.6% in a series of 211 cases); or the milder form may not be present and the subacute may be the first to appear. After a mild onset the temperature rises suddenly on the fourth or fifth day, the glands become enlarged, and a yellowish exudate appears on the tonsils, on the posterior pharyngeal wall, and on the pillars of the fauces. The diphtheric process spreads to the postnasal space, the nasal cavities, and the larynx, and gives rise to ulceration and tissue destruction varying in form and extent. This purulent form of rhinitis is always the result of extension from the postnasal space, and therefore develops a few days later; the clinical picture presents no characteristic features to distinguish it from diphtheric disease of the nose. There is, however, a characteristic discharge of a thin, yellowish, offensive fluid, tinged with blood, from the excoriated nares, which, in connection with the glandular enlargement, is of some value for early diagnosis. It is a sign that the nasopharynx is involved, and appears even before the nose itself is directly attacked.

It is somewhat remarkable that the larynx is rarely involved in this form of the disease, just as in the catarrhal variety, so that a laryngeal stenosis simulating true diphtheria is a rare occurrence. If the membrane does spread to the larynx, it is found to be soft and semifluid, and much less adherent than in diphtheria. In rare cases edema of the larynx and asphyxia were observed; Moure saw an abscess at the base of the epiglottis and about the upper part of the left ventricular band which ruptured spontaneously on the tenth day of scarlet fever.

The loss of substance caused by the destruction of large tracts of mucous membrane in the postnasal space and on the pillars eventually leads to the formation of permanent scars and cicatricial contractions, which in later life may easily be mistaken for syphilitic scars, especially when they occupy the interval between the pillars of the fauces and the posterior pharyngeal wall. The formation of adhesions in the interior of the nose in scarlet fever should also be mentioned; the skeleton itself is never involved.

Finally, Heubner describes a *malignant form* which presents all the symptoms of an intense general septicemia,

with rapid destruction of the mucous membrane of the nose and throat, and with necrosis of the cervical and parotid glands and of the skin covering them; the glands at first are of a stony hardness. This variety, which appeared in 16.3% of the 211 cases in Heubner's clinic, terminated fatally about the seventh to the tenth day.

Diphtheric disease due to scarlet fever presents certain important distinctions from true diphtheria, caused by Löffler's bacillus, in the nature of the sequels which are apt to follow. The peripheral palsies which constitute some of the most dreaded after-effects of diphtheria, and of which we are concerned only with paralysis of the pillars of the fauces and of the larynx, are never observed after scarlet fever. This is confirmed by Heubner and by Leichtenstern, who refers to 600 cases, so that the isolated contradictory cases, cited for the most part by older writers, must be explained in some other way. Without giving the individual case histories, Wreden [1] makes the statement that he observed paralysis of the pillars of the fauces, the vocal cords, the extremities, and the heart in eighteen cases of nasal and pharyngeal diphtheria complicated with scarlatina. But, in the first place, any ulceration in the pillars of the fauces may interfere mechanically with the movements of the uvula; and, in the second place, there have been reported cases of genuine diphtheria combined with scarlet fever when the finding of Löffler's bacillus rendered the diagnosis absolutely certain (Jurgensen[2]). In such cases of double infection the occurrence of post diphtheric palsies is, of course, conceivable, but they must be attributed to the diphtheria and not to the scarlet fever.

Scarlet fever plays a more important rôle in the etiology of *diseases of the ear* than any of the other infectious diseases. The literature does not afford many statistics in regard to the frequency of aural disease as a complication of scarlatina, the only statistics I was able to find being those of Burckhardt-Merian, who reports middle ear disease in 5 out of 15, and in another series in 8 out of 36, cases. On the other hand, the frequency of scarlatina as the original cause of aural diseases forms the subject of numerous articles based on a large amount of material. The most reliable statistics are those contributed by

[1] Wreden, "Mon. f. Ohr.," II, p. 151.
[2] Nothnagel's "Spec. Path. u. Ther.," IV, 2, p. 133.

Bezold,[1] who collected 640 cases of aural disease secondary to scarlet fever, covering a period of eleven years, from 1881 to 1892, in which 984 organs of hearing were affected, one-half of all the cases being bilateral. The total number of cases of scarlet fever during the same period Bezold estimated from other statistical sources at 17,087, so that 3.75 % of all aural affections must be attributed to scarlet fever. This percentage tallies approximately with the results of other statistics,[2] in which the percentage ranges from 2.3 to 9.3, with a total average of 5.17 %. The frequency with which the different parts of the ear are affected varies greatly, affections of the middle ear showing a heavy preponderance over those of the internal, and especially of the external ear, which are extremely rare. To show how frequently the middle ear is involved, it is only necessary to state that about 12.1 %[3] of all cases of purulent otitis media must be regarded as secondary to scarlet fever.

While these figures alone suffice to show the significance of scarlet fever in the etiology of aural diseases, it becomes even more apparent when we consider the functional disturbances and other sequels that follow in its wake. To quote at random from Bezold's statistics, we find the appalling statement that in 109 out of 217 cases of chronic purulent otitis media with polypi, and in 154 out of 315 cases without polypi, the disease lasted longer than eight years.

When it is considered that in 48.5 % of all Bezold's cases the distance at which whispered tones could be heard was less than ½ of a meter, and that in 13.5 % a whisper could not be heard at all, and when, in addition to this, the frequency of acquired deafmutism after scarlet fever, which shows an average of 19 %,[4] is taken into account, it is easy to understand the otologist's repeated appeals to the general practitioner, adjuring him to devote more attention to aural complications in scarlet fever than appears to have been done hitherto.

We will first consider the grave and fortunately rare form of otitis which is designated the diphtheric form, being anal-

[1] "Uberschau über den gegenwärtigen Stand," etc., 1895, Wiesbaden, Bergmann, pp. 168, 169, table VIII.

[2] Blau, "Arch. f. Ohr.," 27, p. 140.

[3] Average of Blau's figures, "Arch. f. Ohr.," 27, p. 142, table II.

[4] Blau, *loc. cit.*, p. 143, table IV.

ogous to diphtheroid scarlatina of the throat. The same confusion that prevailed in regard to the diseases of the throat before the subject was somewhat clarified by the works of Heubner and others still befogs the various descriptions of diphtheric inflammations of the ears in scarlet fever. The opinion is abroad, based chiefly on the writings of Wreden and Burckhardt-Merian, that otitis in the course of scarlet fever in practically every instance consists in a diphtheric inflammation of the middle ear. The 18 cases reported by Wreden, which date from the year 1868, can not be regarded as authentic, as they represent suppurations occurring "during the decline of scarlet fever" (sub decursu febris scarlatinosæ); the time of their appearance (late in the course of the disease), and the statement that they were frequently followed by palsies, arouse the suspicion that we have to deal with a genuine complication of scarlet fever with diphtheria, and Burckhardt-Merian's remarks on diphtheria, found in his paper on otitis in scarlet fever, are of no value in the present discussion, for the very reason that the diphtheria of scarlet fever is a very different thing from genuine diphtheria.

From the description [1] [2] by Moos and Pulitzer, who designate diseases of the ear in scarlet fever simply as diphtheric diseases or scarlatino-diphtheric suppurations of the middle ear, it might be inferred that the diphtheric form is the only possible aural complication in scarlet fever. But how, then, are we to reconcile the frequency of middle ear disease in scarlatina with the rarity of diphtheric disease of the middle ear? Gottstein's unsuccessful attempt to prove that the diphtheric aural affection forms an integral part of the morbid process in scarlet fever is followed by the ingenious explanation that the aural affections did not come under the observation of the ear specialist until after the end of the diphtheric and the beginning of the purulent stage, while Wreden had the opportunity to observe the disease in its early stages. A strange caprice of fate, indeed, if that early diphtheric stage regularly escaped the notice of the physician!

The most authentic cases of a diphtheric form of otitis

[1] "Schwartze's Handb.," vol. I, "Allgemeine Aetiologie der Ohrenkrankheiten."

[2] "Lehrb. der Ohrenheilkunde."

media in scarlet fever are those reported by Blau,[1] Katz,[2] and Siebenmann.[3]

According to these observers, the clinical course of the disease is as follows: Coincident with the diphtheric complication in the throat there takes place a rapid destruction of the tympanic membrane followed by an otorrhea, in which the fluid is described as muddy and serous in character, not purulent. A diphtheric membrane is formed on the mucous membrane of the tympanum and discharged into the external meatus. The diphtheric process is not limited to the middle ear, and may manifest itself in the formation of membranes in the external meatus and in the auricle, as was observed in several instances. Blau's case was not followed to the end; the cases reported by Katz and Siebenmann terminated fatally on the fifteenth and twentieth day respectively. Siebenmann's attempts to discover diphtheria bacilli in the membranes found in the middle ear after death were unsuccessful.

As the disease progresses the mucous membrane undergoes necrosis; the bones of the tympanum are laid bare, and may eventually become carious. The serous, muddy secretion is later replaced by a purulent discharge; in other words, the diphtheric process is converted into a chronic suppuration characterized by extensive carious destruction. The coincidence of this form of otitis with the diphtheric process in the throat suggests the thought that they are both caused by the same malign influence manifesting itself in different parts of the body. The simplest explanation of the aural complication on this theory would be direct extension of the diphtheric process in the pharynx through the Eustachian tube, but of this we have no proof. In Siebenmann's case, which is so excellently described, the tube was unfortunately destroyed at the autopsy.

A much more frequent form of aural complication in scarlet fever is *acute otitis media* without any special characteristic features. It begins during the period of desquamation,—that is to say, in the third or fourth week of the disease,—with a rise in temperature and pain radiating from the affected ear and increasing in severity toward evening, so that the patient is unable to sleep. There is usually

[1] "Berlin. klin. Wochen.," 1881, Nos. 49, 50.
[2] "Berlin. klin. Wochen.," 1884, No. 13.
[3] "Zeitschr. f. Ohr.," XX, p. 1.

some glandular enlargement behind and under the angle of the jaw, on the mastoid process, or in the back of the neck. The tympanic membrane is red, swollen, and inflamed, and bulges so that immediate paracentesis is indicated ; if it is not performed, spontaneous perforation takes place, often within a few hours after the first appearance of subjective symptoms. An important variation from the ordinary clinical picture as just described is to be found in the description given by some observers of a remarkable absence of pain, which they arbitrarily attribute to anesthesia of the sensory nerves.

Up to this point the course of the disease is essentially the same as that of simple otitis media, but after the occurrence of perforation, which preferably takes place in the lower anterior quadrant, the membrane undergoes rapid disintegration and is often totally destroyed. According to Bezold,[1] total destruction of the membrane occurs in 25.2% of all cases of scarlet fever, and a destruction of at least two-thirds of the disc in 24.7%. The flow is very abundant and presents the usual mucopurulent appearance. The most characteristic features of the otitis media are an obstinate resistance to treatment and a tendency to carious destruction, which frequently involves the ossicles, as well as the bony walls of the tympanum and contiguous cavities. As the hearing is much impaired in scarlet fever, it is probable that the disease extends to the internal ear ; but whether we have to deal simply with a secondary carious destruction of the labyrinth, or with a special localization of the disease, is not known.

How are we to explain the *origin of this form of otitis in scarlet fever?*

Is it a disease due to the extension of the initial pharyngitis through the tubes, and presenting phenomena in the form of an otitis media such as we must expect after any catarrhal rhinopharyngitis? or is it a specific disease caused by the virus of scarlet fever or by certain toxins which it produces?

Mere hypotheses add little to our knowledge, which must necessarily remain incomplete as long as the nature of the scarlatinal contagium is unknown. Certain conclusions can, however, be drawn as to the origin of the

[1] *Loc. cit.*, p. 172.

disease from the time of its appearance as a complication and from its general character. It occurs regularly during the period of desquamation—at a time, therefore, when there exists a tendency to other complications as well; for, except in the rare cases of diphtheroid scarlatina, there is no record of its occurring immediately subsequent to the scarlatinal angina which lasts only a few days. That the resisting power of the mucous membrane of the tubes and of the tympanum to the invasion of pathogenic germs—which might set up a suppurative process in the middle ear independent of the scarlatina—is especially lowered during this period of the disease is not only not proved, but is even improbable, as no such condition of affairs is observed in the mucous membranes of the upper air-passages, where secondary streptococcal infections usually follow immediately after the scarlatinal angina, in the first week of the disease.

Again, if we assume that the aural complication is merely accidental, or that it is dependent on the pharyngeal condition, it is, to say the least, remarkable that simple catarrh and mild otitis media without perforation do not occur in scarlet fever, or at least are so rare that they can not be included among the list of complications of the disease.

If, on the other hand, we consider that it is during the desquamation period that we find nephritis,—a disease which is unquestionably toxic in character and therefore indicates a septic condition of the organism,—the assumption that the aural complication is due to the action of the same toxins seems plausible. If a parallel could be established between nephritis and purulent otitis media,—as in a case observed by Voss, where the onset, course, and subsidence of the two diseases progressed *pari passu*,—it would offer another argument in support of the dependence of the aural disease on a general intoxication of the system.

The bacteriology of scarlatinal otitis media and the significance of a mixed infection have not as yet been discovered.

3. VARICELLA.

In varicella, vesicles appear on the mucous membrane of the mouth and pharynx at the same time as the skin eruption; in rare cases a few isolated pustules were found in

the larynx. Cases of grave suffocative laryngitis have been described by Marfan and Hallé [1] and by Harlez,[2] which, it appears, occurred suddenly at the time of the eruption, with symptoms of asphyxia, attended with hoarseness, cough, and muffled phonation. Tracheotomy was required in every instance; no laryngoscopic examinations are reported; in one of the cases ulcers were found on the vocal cords at the autopsy.

In a unique case reported by Bürkner,[3] two pustules were found in the external auditory meatus, with only a scanty eruption on the scalp.

4. VARIOLA.

In variola the mucous membranes contiguous to the external skin are regularly attacked. E. Wagner [4] found that the nasal mucous membrane was affected in every case in which it was examined. In a series of 170 cases the upper pharynx alone was affected twice; the pharynx and larynx alone, 38 times; the pharynx, larynx, and upper half of the trachea, 54 times; the pharynx, larynx, trachea, and large bronchi, 52 times. The larynx was therefore involved altogether in 144 cases out of the 170. Between the third and sixth day of the smallpox eruption (Mackenzie) pustules make their appearance in the pharynx and spread to the postnasal space and larynx. They may be isolated in different parts of the larynx, or they may be multiple and coalesce to form large ulcers. At first the pustules resemble those on the external skin, but the covering of mucous membrane soon becomes macerated, is cast off, and leaves a red, excoriated patch, which is apt to bleed. In the hemorrhagic form ecchymoses appear in the mucous membranes. These superficial eruptions on the mucous membranes are complicated with deeper ulcerative processes, which lead to edema of the larynx and abscess formation; by extension to the cartilages this may give rise to a perichondritis of the larynx, as illustrated by

[1] "Rev. d. mal. d. l' enf," XIV, Jan., 1896, rep. in "Semon's Centralb.," XII, p. 499.

[2] "Indép. méd.," July 14, 1897, rep. in "Semon's Centralb.," XIV, p. 214.

[3] "Arch. f. Ohr.," 18, p. 300.

[4] "Arch. d. Heilkunde," XIII.

Türck [1] in a number of cases. In addition to the pustular, Mackenzie mentions a papular form, and Löri reports hyperemia of the mucous membranes without pustular eruption.

Rühle,[2] among others, speaks of a diphtheric croupous inflammation of the laryngeal mucous membrane, with secondary invasion of the postnasal space. That this was the result of confluence of the pustules is denied by Löri on the ground that there never were any pustules on the mucous membrane; but E. Wagner says that in the numerous cadavers he examined the pustules were often so closely set, especially in advanced stages of the disease, that it was difficult to demonstrate their variolous character. Finally, we have the occurrence of *palsies* as a very rare complication. Mackenzie saw two cases which were followed by paralysis of the adductors of one cord. The nature of these palsies is not known, but they are probably due to mechanical causes, such as ankylosis of the arytenoid cartilage, observed after perichondritis or after the cicatrization of a deep ulcer.

Aural disease during smallpox was studied by Wendt[3] in 168 organs taken from 84 persons of all ages who had died in various stages of the disease. As the ears were found to be intact in only 3 cases, there can be no doubt of the frequency of aural complications in variola. The nature of the lesions varies, according to Wendt's findings; in some instances the morbid process was identical with, or closely related to, variola, in others the lesions were such as occur in connection with other constitutional or local diseases, or even without them. From the external skin the eruption spreads to the concha and auditory meatus; from the mucous membrane of the pharynx to the pharyngeal orifice of the tubes. Whether the epithelial thickening and suppuration, and the hyperemias, hemorrhages, and exudations in the middle ear, are the product of the primary disease or the result of the tubal condition is an open question.

So far as has been observed, the tympanic membrane is never the seat of a pustular eruption, but it is frequently found to be red and swollen. These anatomic findings

[1] "Klinik der Kehlkopf krankheiten."
[2] "Klinik der Kehlkopf krankheiten," 1861.
[3] "Arch. f. Heilkunde," XIII.

of Wendt are directly contradicted by the clinical observations of Ogston.[1] The latter, after examining the ears of 229 smallpox patients, reached the conviction that "the structures and tissues of the ear itself are not affected by variola."

The prognosis, according to Wendt, is favorable; he believes that the healing of the smallpox lesions in the ear is not followed by any functional disturbance, nor have there ever been found cicatricial stenoses or synechiæ from the healing of pustules in the external auditory meatus or in the tubes.

5. TYPHOID FEVER.

The laryngeal phenomena occurring in the course of typhoid fever may be divided into three main groups—catarrhal conditions, ulcerations, and palsies; edema and perichondritis are regarded as accompaniments or complications of one of the three main divisions. There are plenty of data to determine the frequency of these complications, but a certain reserve is necessary in drawing general conclusions, for the statistics would be quite different if a systematic laryngoscopic examination were made in every case of typhoid fever, without waiting for the patient to complain of pain in the throat or for the appearance of such obvious symptoms as dyspnea and aphonia. The results obtained vary according as they are based on observations made on the living subject or on the cadaver, for complications are naturally much more frequent in severe cases of typhoid terminating fatally than in the milder forms. Another factor is the severity of the epidemic that happens to furnish the basis for the statistics. The most comprehensive figures are those published by Lüning,[2] who puts the percentage, as computed from clinical statistics, at 3, and the postmortem percentage at 17.

It would be interesting to know the relative frequency of the various forms of laryngeal disease; but on this point we can not hope for any information from the results obtained at autopsies, as they naturally include only the gravest complications, such as perichondritis or diphtheric disease.

[1] "Arch f. Ohr.," VI, p. 267. [2] "Langenbeck's Arch.," vol. XXX.

Clinically speaking, simple catarrh and superficial ulceration are the complications most frequently observed, while deep ulcerations which lead to edema and perichondritis, or which, when extensive, present the so-called diphtheric form ("laryngotyphus") are much rarer. If the latter are more frequently and more fully described it is only because of their alarming symptoms and the laryngeal stenosis which characterizes them and directly threatens the patient's life. Stenosis and edema of the larynx are sometimes induced by typhoid processes in neighboring organs; thus, cases have been reported in which acute inflammation or abscess formation in the thyroid gland—which condition appears to be quite frequent in the course of typhoid—led to compression of the trachea and edema of the larynx. Our knowledge of post-typhoidal palsies is of very recent date. They were formerly considered a very rare complication, for Lublinski could collect no more than 25 cases, including 6 of his own, and Landgraf met with only 2 cases of laryngeal palsies among 166 typhoid patients. A special interest, therefore, attaches to Przedborski's[1] report, accompanied by very complete case histories, of 25 laryngeal palsies among 100 cases of abdominal typhoid, and of 7 among 25 cases of exanthematous typhoid.

The pharyngeal and laryngeal mucous membrane is often attacked by catarrh in the beginning of the disease, while, on the other hand, the nasal mucous membrane not only escapes but presents an unusually dry appearance. The only nasal symptom observed is epistaxis. The hemorrhage shows a predilection for the septum, but is also observed in other parts of the mucous membrane. In a few cases which came under my observation the nasal mucous membrane after the hemorrhage presented the previously mentioned desiccated appearance, the septum was marked with rhagades, while the walls and interior of the nose were covered with larger and smaller masses of black, clotted blood, which moved to and fro with the respiratory movements. The epistaxis occurs in the beginning of the disease. As the patients at this time are usually in bed and more or less prostrated by the fever, the blood usually flows backward into the throat, and the resulting bloody sputum may give rise to errors in diagnosis. Perforation of

[1] Volkmann's "Sammlung klin. Vortr.," No. 182, 1897.

the septum, like that produced by a perforating ulcer, has been observed after typhoid fever. Typhoid pharyngitis and laryngitis are characterized by intense redness, while the swelling of the mucous membrane is comparatively slight. Marked swelling and edema are rare in this stage. The so-called catarrhal redness of the larynx in typhoid is not uniformly distributed. It may be due to venous stasis (Landgraf[1]).

Ulcers appear in various forms both clinically and anatomically. The commonest variety consists in superficial ulcerations from necrosis of circumscribed portions of the swollen mucous membrane. They manifest a preference for certain regions of the pharnyx and larynx, being found almost regularly on the faucial pillars, the free border or laryngeal surface of the epiglottis, the aryepiglottic folds, and occasionally below the glottis; they are rarely seen on the vocal cords. At first there is a diffuse catarrh, and the mucous membrane is darker in color and slightly swollen in the areas mentioned; the epithelium soon breaks down, and exposes a small, shallow ulcer with a yellowish floor, resembling herpes; similar ulcers appear in the neighborhood and coalesce with the original one to form larger, irregular, quite superficial ulcers, with clearly defined edges, but without redness or swelling of the adjacent parts. These ulcers occur in all stages of typhoid, and may be due to a variety of causes. They can not be regarded as decubital ulcers,[2] as there is no reason why, if we accept such an etiology, similar ulcers should not occur in any other disease attended with the same degree of prostration; nor can they be attributed to the effect of contact and direct infection with the typhoid bacillus, as it has been possible in only a very few instances to demonstrate the presence of bacilli in the secretion, and there is no satisfactory explanation of the mode of infection.

The ulcers are undoubtedly to be regarded as the result of a nutritive disturbance in the catarrhal mucous membrane connected with the general typhoidal infection, but their mode of origin and direct dependence on infection by the bacillus are not so clear. They are superficial, and, on the whole, may be considered benign, as they heal without leaving a scar and do not require any local treat-

[1] Landgraf, "Charité Ann.," 1889.
[2] Rühle, "Verhdl. der Naturf. Vers.," 1862.

ment. There are cases, however, in which the ulcers extend to the deeper structures, probably as the result of a mixed infection. Eppinger[1] calls them mycotic necrotic ulcers, and gives a detailed description of the way in which they invade the deeper structures and eventually destroy the perichondrium and cartilage. The cases which go on to phlegmon formation are to be explained as due to such mixed infection; Villecourt[2] describes one that was localized in the glottis and posterior laryngeal wall. These ulcers differ both clinically and anatomically from the alterations described by Eppinger under the name of diffuse typhoid infiltrations; he considers them in every way analogous to the typhoid lesion in the intestinal follicles, and therefore assumes that they originate in circumscribed areas containing adenoid tissue in the mucous membranes of the upper air-passages.

These infiltrations lead to ulceration, the ulcers being distinguished from those of the first group by the hardness and swelling of their undermined edges. Although they show no tendency to invade deeper structures, they may, as the result of a mixed infection, assume larger proportions and lead to diseases of the cartilaginous structure of the larynx.

There is one form of diphtheria accompanying typhoid often described by older authors (Landgraf also mentions a case of typhoid which was probably complicated with true diphtheria), in which the disease is said to originate in the larynx and pharynx and to extend upward to the nasal mucous membrane. As true diphtheric membrane corresponding to casts of the interior of the larynx were observed, the occurrence of such cases can not well be doubted, though they have never been seen by later observers, such as Schrötter, for instance. At all events, these cases do not represent a true diphtheria, but rather the last group of typhoid ulcers, in which, as a result of mixed infection, croupous processes develop.

As in all forms of ulcerations which occasion destruction in the deeper tissues, the healing of the ulcers leaves defects and adhesions, which often lead to stenosis of the larynx and may subsequently require local treatment. Such sequels may be of various kinds; their diagnosis

[1] Klebs, "Handb. d. pathol. Anatomie," vol. II, Abth 1.
[2] "Gaz. des hôp.," 1893, No. 116.

often presents great difficulties, and they may be confounded with syphilitic, diphtheric, and other scars, for post-typhoid adhesions present no special characteristics. Thus, Halász[1] described a case of membranous adhesions between the lower edges of the vocal cords after typhoid.

Diseases of the perichondrium and of the cartilages of the larynx after typhoid fever deserve special attention. They are always to be regarded as secondary, due to the extension of the ulcerating process to the perichondrium. They present various clinical appearances, and closely correspond with diseases of the cartilage from other causes; a large number of very instructive cases, in part illustrated with excellent cuts, are found in Türck's text-book. The various cartilages may become diseased singly or in connection with others; according to a statistical investigation of the frequency in the individual cartilages (by Lüning, Bussenius, and others), the cricoid cartilage is attacked far more frequently than any other. Lüning found it affected in 44 out of 55 cases of perichondritis; Bussenius[2] in 49 out of 72 cases. This phenomenon is worthy of special attention, as Bussenius has shown that the distribution of the disease in syphilis and tuberculosis is quite different as regards the individual cartilages, the arytenoid cartilage being affected in by far the greater number of cases.

A few cases of paralysis of the laryngeal muscles have been observed. They occur chiefly in the stage of convalescence (Mendel, Boulay), but may also be met with, according to Przedborski, in the febrile stage. The latter is, in fact, said to be the rule in typhus exanthematosus (petechial typhus). The paralysis presents no characteristic type, and all the muscles may be affected, either singly or combined; Mendel and Boulay found paralysis of the adductors in only 4 out of 17 cases. The abductors must be regarded as most frequently affected by paralysis, but Przedborski, in his 32 observations, reached a different conclusion, finding both abductors and adductors affected with about equal frequency. But as the former include a number of paralyses of the vocal cords, such as we frequently observe in anemic persons after exhaustive infec-

[1] "Pest. med. chir. Presse," 1893, No. 40; see "Centralb. f. klin Med.," 1893, No. 52.
[2] "Charité Ann.," 1896.

tious diseases, and are therefore in no sense peculiar to typhoid, it is quite possible that the figures may have to be again revised. It would be interesting to investigate the fact reported by Przedborski that the muscles become affected one after the other without any definite order such as is usually observed in the development of a recurrent paralysis. Opinions are divided as to the nature of the paralysis, but the general tendency is to regard it as analogous to that which occurs in diphtheria; in other words, as a peripheral paralysis, such as is observed in other infectious diseases, although it is still a matter of dispute whether the muscles themselves or the peripheral nerves suffer a pathologic alteration. The attempt has also been made to explain it as a central paralysis due to hemorrhage in the central organs. According to Przedborski, the prognosis as to recovery is favorable, as he found that the paralysis usually disappeared in the course of from one to three weeks.

In a few cases a simultaneous paralysis of one-half of the uvula was observed—paralysis pharyngoglossolabialis; as, for instance, in one case of a boy twelve years old.[1]

The ear frequently becomes involved in typhoid, the complications being more frequent in typhus exanthematosus (petechial typhus) than in typhus abdominalis. We possess a few statistics concerning their frequency, based on a number of examinations which were made on a series of typhoid patients without regard to the presence of any subjective symptoms. Bezold [2] found fifty aural complications among 1243 cases of typhoid (4.02 %); Hengst,[3] 28 among 1228 (2.3 %); Botkin [4] saw 19 cases of purulent otitis among 357 typhoid patients. The statistics of Zaufal, Kramer, and Schmalz, quoted by Bürkner, yield a percentage which varies from 1.8 to 2.5. These figures do not, as Haug seems to imply, relate to the frequency of otitis in typhoid fever, but to the frequency with which typhoid fever was given as the cause of aural disease. The complications consist mainly in disease of the middle ear; a few isolated cases have also been reported of involvement of the external and internal ear, but of this we know very

[1] Brück, "Sest. med. chir. Presse," 1891, No. 30; see "Semon's Centralbl.," VIII, p. 510.

[2] "Arch. f. Ohr," XXI.

[3] "Zeitschr. f. Ohr.," XXIX, p. 184.

[4] See "Mon. f. Ohr.," 1895, p. 135.

little. We will first discuss the *middle ear diseases* in typhoid. Among the 50 cases observed by Bezold, the middle ear complication in 48 consisted of inflammation, while in only 2 cases was there a simple tubular catarrh which was not dependent on the typhoid fever. The inflammatory phenomena usually appear in the fourth or fifth week of the disease (according to Bezold, 45 times between the twenty-fourth and twenty-fifth day, and only 5 times before the twentieth day); they are heralded by rises in the temperature, which can be referred to the typhoid disease, although they occur in the stage of recrudescence, in which the fever shows a remittent type with occasional marked exacerbations. The patients usually complain of earache and tinnitus aurium, and the attendants note a diminution in their power of hearing.

The course of the otitis media is variable, and three forms may be distinguished: a simple inflammatory form without perforation, a purulent form with perforation of the ear-drum, and a form in which involvement of the mastoid process is the prominent feature.

In the first form the otoscopic picture shows moderate redness of the ear-drum, especially in the region of the handle of the malleus, without any bulging of the membrane. According to Bezold, the congestion evinces a marked tendency to spread to the external meatus. The ear-drum shows little or no bulging, and, as a rule, is not swollen. This form, which is the mildest, may pass into the purulent perforative variety, or the inflammation is so acute from the outset that the ear-drum, which shows a marked redness, soon bulges outward, and perforation rapidly takes place. The suppuration itself is not characteristic; perforation is said to occur preferably in the posterior inferior quadrant. The size of the opening varies, and cases of multiple perforation have even been reported. I myself once observed a case in which bilateral chronic suppuration, which had existed before the onset of the typhoid disease, became arrested during the fever. In the fourth week earache made its appearance. The ear-drums on both sides were very red, and on the left side there was a defect, but no discharge, while the fever still continued high. Eight days later, after the fever had fallen, the congestion subsided, and marked suppuration again set in. These forms of otorrhea which subside during high

temperatures were referred to in the discussion of croupous pneumonia, and appear to occur in all infectious fevers where the patient is a subject of chronic suppuration.

One peculiarity of purulent otitis media during typhoid, which is mentioned by most authors, is the early involvement of the mastoid process. Inflammatory phenomena make their appearance in the mastoid process at the same time that the acute inflammation invades the middle ear, and various cases have been described in which there was marked tenderness on pressure out of all proportion to the appearance of the ear-drum. Brieger observed a case in the eighth week of typhoid in which fluctuation was made out over the mastoid process within four days after the first appearance of the earache, while the corresponding ear-drum was markedly hyperemic and quite flat, and only ruptured on the next day, the perforation being very small and followed by a slight discharge. An operation was performed a week after the onset of the pain, and showed the presence of sequestrums in the mastoid process. The case ended fatally in five weeks, death being due to thrombosis of a sinus. Brieger points out that this does not correspond to the ordinary course of bone disease following typhoid, as there is usually a tendency to spontaneous cure of the inflammation. There is no doubt that the bone is extensively involved. This is shown by Bezold's investigations; in 19 out of 41 cases he found marked tenderness on pressure, which in 11 cases made its appearance at the same time as the inflammation. In 5 out of these 19 cases a periosteal abscess resulted, and required incision.

It being established that the bone disease either progresses *pari passu* with the otitis media or precedes it, the question of the etiologic relations existing between the bone disease and typhoid otitis now presents itself. According to Bezold, the inflammation in the middle ear may begin in one of the three following ways:

First, by direct extension of the inflammation from the nasopharynx through the tube, simple occlusion of the tube being probably insufficient to be regarded as an etiologic factor, at least for the suppurative processes.

Second, by the passage of septic material directly from the nasopharynx into the middle ear.

Third, by the formation of emboli in the vessels of the mucous membrane of the middle ear, emanating either

from an endocarditis and thrombosis of the left heart, or from purulent foci in the periphery.

Bezold therefore considers the aural complication as secondary and excludes the effect of the general infection as an etiologic factor. If Bezold's exposition of the etiology is accepted, it is difficult to explain how the disease, which is at first localized in the middle ear, can be transplanted to the walls of the mastoid process with such rapidity as to make the secondary, appear to precede the primary disease. Even if we admit the possibility of the middle ear becoming infected through the tubes, we can not discard the theory that we have to deal with an acute osteomyelitis of the mastoid process, which is to be regarded as a true complication of the typhoid disease. The demonstration of typhoid bacilli would settle the matter beyond dispute; unfortunately, we do not possess any bacteriologic data; however, the course of the bone disease, as has been previously stated, is in itself quite different from that which is usually observed in the complications of typhoid fever, and even if we assume a mixed infection to explain the sequestration of the bone and the formation of periosteal abscesses, the question why the disease in the bone should precede or even accompany the suppuration from the ear remains unsolved.

Complication of the external ear (the auricle and the external meatus) is a very rare occurrence. Haug [1] quotes a case of gangrene of the auricles from Obre. Von Tröltsch and Hoffmann [2] each observed a case of suppuration of the parotid gland with rupture into the external meatus. In Hoffmann's case there was a fistula at the junction between the cartilaginous and bony portions of the meatus. On the other hand, Botkin [3] observed bilateral otitis externa 21 times among 26 typhoid patients, and erects the improbable hypothesis that suppurations from the middle ear in typhoid are due to an extension of otitis externa to the ear-drum and to the tympanic cavity.

An apparent reduction in the power of hearing is frequently met with in the course of typhoid fever, although no objective changes can be found to account for it. It is quite unjustifiable to interpret such cases as nervous

[1] "Die Krankh. des Ohres," etc., p. 90.
[2] "Arch. f. Ohr.," IV, 6th Observation.
[3] See "Mon. f. Ohr.," 1895, p. 135.

deafness, for clinical experience teaches that the difficult hearing is due to somnolence, and improves as soon as the mental faculties are restored. I have myself observed that the hearing varies during the febrile stage, being remarkably improved during the remissions of the temperature which follow cold baths. When Haug [1] remarks "that this typhoidal deafness sometimes reaches its highest point at the crisis of the general disease, and then gradually diminishes and allows the ear to return to its normal condition during the stage of convalescence," and insists particularly on the fact that "disturbances of the sphere of coordination have never been observed," we may be pardoned for expressing a doubt of this "nervous ear affection."

This must not, however, be taken to imply that we deny the possibility of the nervous hearing apparatus being involved in typhoid fever, and there are, in fact, a few observations which prove that difficult hearing and tinnitus aurium, with the other phenomena of the nervous affection, undoubtedly occur during the stage of convalescence; in fact, the anatomic investigations of Pulitzer, Moos, Lucae, and Schwartze demonstrated an anatomic basis for this clinical picture—a hyperemia of the internal ear or ecchymoses and hemorrhages in the vestibule and in the cochlea. The clinical cases of nervous deafness which have been described as progressive after typhoid fever, must, in the absence of detailed histories, be accepted with a reservation, as they may have something to do with the exhibition of quinin or salicylic acid during the course of the fever.

6. INFLUENZA.

Although the port of entry for the carriers of the infection of influenza is probably to be sought in the mucous membranes of the upper air-passages, the parts themselves are directly involved in only a small percentage of the cases. Leichtenstern [2] has designated this form as catarrhal respiratory influenza, in contradistinction to the gastro-intestinal form and the purely toxic form with fever and nervous phenomena. The frequency of rhinitis is variously given at from 25% to 79%, that of laryngitis from 5% to 16%; these figures appear remarkably low in comparison with the

[1] *Loc. cit.*, p. 95.
[2] Nothnagel's spec. "Path. u. Ther.," vol. IV, I, p. 77.

frequency with which these conditions are observed in practice.

There can not be said to be a typical clinical picture for the complications of influenza in the upper air-passages, for they manifest themselves under the most various forms. Two principal groups are distinguished—one affecting principally the mucous membrane, the other the nervous system. With regard to affections of the mucous membranes, it has been pointed out by Leichtenstern that the inflammation is not uniformly distributed over all the mucous membranes, and that the deeper portions do not always become affected secondarily to the disease in the upper portions,—*i. e.*, the nose and the nasopharynx, as is the case in most other conditions,—but every portion of the respiratory tract is capable of becoming primarily affected by the morbid process.

In the nose the inflammation presents the picture of an acute rhinitis which is distinguished from an ordinary coryza only by the rapidity of its course, the inflammatory symptoms and secretion subsiding within a very few days. The rhinitis is occasionally accompanied by epistaxis, although we find very contradictory statements in regard to this symptom. Schmidt and Litten regard epistaxis as a very frequent complication, while Tissier,[1] Leichtenstern, and Fränkel,[2] on the other hand, say that it is comparatively rare. We should mention the occurrence of acute or, later, chronic suppurations in the accessory cavities as one of the complications. Thus, the maxillary sinus is frequently the seat of an acute inflammation, accompanied with nasal obstruction and facial neuralgia, which immediately disappears either spontaneously or after the swelling in the mucous membrane has subsided and the orifice of the cavity has been exposed. The best descriptions of suppurations of the accessory cavities are given by Tissier, who claims to have found all the various sinuses affected. Ewald[3] reports a very malignant case in which a purulent basal meningitis developed after an empyema of the antrum of Highmore had been opened; the meningeal complication at the autopsy was accounted for by the finding of a suppuration in the ethmoid cells.

[1] "Ann. des mal. de l'oreille," 1892, p. 425.
[2] "Semon's Centralbl.," VII, p. 38.
[3] "Berlin. klin. Wochen.," 1890, No. 3.

Catarrh of the pharynx and larynx also presents the ordinary picture of an acute inflammation, except that hemorrhage appears to be a more frequent complication than in the nose; the term *laryngitis hemorrhagica* has been applied to this form of the disease. The affected mucous membranes are frequently the seat of whitish patches, not elevated above the swollen and reddened mucous membrane. They are analogous to similar patches found in acute catarrh, and are to be interpreted as a superficial necrosis. In a few instances marked edema of the laryngeal mucous membrane was observed, which even went on to abscess formation, and Rethi described a coexisting perichondritis of both plates of the thyroid cartilage.[1]

As regards nervous diseases, a few cases of anosmia and parosmia have been reported, and while paralysis of the palatal muscles and of the constrictors of the pharynx may occur, by far the most important complication consists in paralysis of the laryngeal nerves, which must be regarded as a typical influenza neuritis such as occurs in all parts of the body. Besides rare cases of paralysis of the sensory superior laryngeal nerve we meet with paralyses of the laryngeal muscles, both of the adductors (Onodi saw an isolated paralysis of the cricoarytenoideus lateralis, and Rosenberg frequently noticed paralyses of the vocal cords) and of the abductors; they usually make their appearance after the acute inflammation has subsided. So far as the observations have gone, the abductors appear to be more frequently involved than the adductors, and both unilateral and bilateral paralysis of the crico-arytenoideus posticus has been observed. Seifert[2] reports a unique case of a right-sided total paralysis of the vagus which he regards as peripheral in origin. Besides the usual cardiac and circulatory symptoms there was paralysis of the right recurrent and of the superior laryngeal nerves.

AURAL COMPLICATIONS IN INFLUENZA.

Soon after the appearance of the influenza epidemic of 1889–1890 the attention of aural surgeons was directed to the frequency of purulent otitis media as a complication of influenza, and the numerous observations that have been

[1] "Wien. klin. Wochen," 1894, No. 48.
[2] "Rev. hebd. de lar., d'ot. et de rhin.," 1896, p. 1537.

made since then, and that any physician can make for himself even now in the sporadic cases of influenza, justify the conclusion that this epidemic infectious disease occupies an important place in the etiology of aural complications. It was learned by the statistics of Ludwig and Jansen that a rapid increase in middle-ear diseases occurred during the months of November and December, 1889, and January, 1890, and this increase was attributed to the epidemic which was prevalent at that time. It is important to note that the increase did not affect middle-ear diseases in general, but was limited exclusively to inflammations of the middle ear. Thus, in the Halle Ear Clinic the number reached 137 during the months of the epidemic, as against 41 or 44 during the same months of the preceding years; and, according to Gruber, there were 625 cases from November, 1889, to January, 1890, as against 238 and 84 during the same period of the preceding years. Jansen's statistics are most convincing in this respect: they show that the percentage of acute inflammations of the middle ear, which in the first eleven months of the year 1889 amounted to from 10% to 17.7%, rose to 37% in December, 1889, 29% in January, and 20.6% in February 1890, although there was no appreciable increase in the frequency of simple catarrh of the middle ear. The discrepancy can not be explained as an ordinary increase in the frequency of the disease due to the season of the year, since the comparison with the months of November, December, and January of the five preceding years shows a percentage ranging from 8.1 to 21.5, and in only one winter a percentage as high as 25.5. In spite of the increase in this particular form of disease the total number of patients was not appreciably increased, as might have been expected from the general prevalence of disease during the epidemic. Leichtenstern's objection, that "the statistics of specialists merely show the enormous distribution of the influenza," is quite irrelevant. On the contrary, if we examine the statistics of specialists, we find that the great frequency of certain ear diseases—such as acute inflammation and suppuration of the middle ear which are known to follow in the wake of other infectious diseases—and their abnormally rapid and malignant course during an epidemic of influenza, are not merely accidental, but directly dependent on the epidemic.

With regard to the frequency of aural complications of

influenza in general we possess only general statistics, according to which from 0.5% to 2% of all cases are complicated with disease of the ear; but these figures are probably below the true percentage, as the milder cases of influenza remain only a short time in the hospital, and the aural disease therefore appears only as a sequel.

The otitis in influenza makes its appearance in the form of an acute suppuration of the middle ear from a few days to several weeks after the beginning of the primary disease. As influenza is an infectious disease with a special preference for the upper air-passages, it is probable that a large proportion of the aural affections are due to infection from the nasopharynx through the tubes, and, as such, appear under the form of an ordinary purulent otitis media. There is, in addition, another manifestation of influenza which possesses a distinct hemorrhagic character, and is by many regarded as a pure form of influenza otitis. These two varieties can not be accurately distinguished in practice, as the typical appearance in the latter form disappears after the first few days and is replaced by the picture of an ordinary otitis media. The finding of the bacillus of influenza—which was first positively reported by Scheibe, and after him by several other investigators, although never with any regularity—is of very little importance, as sooner or later in any form of suppuration from the middle ear there develops a mixed infection in which other microorganisms may supplant the primary disease germ.

As regards the clinical course of influenza otitis, it was formerly universally believed that hemorrhages were to be regarded as a regular symptom of the disease in the acute form, in accordance with the first descriptions given by Patrzek, Schwabach, Dreyfuss, and Jankau; Schwartze, however, adheres to his opinion that the hemorrhages are not observed with any greater frequency than in inflammations from other causes. We find ecchymoses, varying from the size of a pinhead to that of a split pea, either single or multiple, on the ear-drum and on the walls of the external meatus; or we may have bluish-red extravasations of varying extent, sometimes covering the entire ear-drum. Körner[1] speaks of secondary circular hemorrhages which he saw through the ear-drum after hypertrophy of the

[1] "Zeitschr. f. Ohr.," XXVII, p. 11.

mucous membrane. The hemorrhages often take the form of villous or pouch-shaped diverticula in the tympanic mucous membrane, due to marked swelling of the mucous membrane of the middle ear, and, after perforation, prolapse through that structure into the external meatus. They show a special tendency to recurrence, and frequently reform after simple cauterization. Some observers speak of perforation taking place in a definite portion of the ear-drum, but the statements are so contradictory that it is not worth while to repeat them; isolated involvement of the cupola (infundibulum cochleæ) in influenza, mentioned by Kosegarten and Haug, must be very rare. The discharges are bloody on the first day, and hemorrhages may occur even later without leading to suppuration, while in other cases the bloody discharge is replaced by serosanguineous fluid, which is eventually followed by suppuration.

The statement that purulent otitis media in influenza is more severe than other forms of suppuration from the middle ear is based on the frequent implication of the mastoid process (according to Jansen, in 57 out of 105 cases, 25 of which necessitated trephining). The complication leads to suppurations in the bone and to periosteal abscess, which are greatly to be dreaded on account of the intensity of the process and its rapid extension. According to Körner, Eulenstein, and Lemcke, primary myelitis of the mastoid process with secondary involvement of the middle ear may occur; but the opposite direction, from the middle ear to the mastoid process, is probably to be regarded as the regular mode of infection.

The internal ear is very rarely involved, and the nature of the condition is not known. Lannois [1] and Barnick [2] described cases of labyrinthine deafness after influenza. According to the former, the prognosis as regards restoration of the hearing is bad; according to the latter, favorable. Gradenigo [3] mentions difficult hearing after influenza, which he interprets as a neuritis of the auditory nerve.[4]

The occurrence of otalgia tympanica is occasionally mentioned, and although the condition can hardly be diagnosed

[1] "Rev. de lar., d'ot. et de rhin.," 1890, No. 17.
[2] "Arch. f. Ohr.," 38, p. 183. [3] See "Arch. f. Ohr.," 36, p. 141.
[4] Comp. Leyden and Guttmann, "Die Influenzaepidemie," Wiesbaden, 1892, p. 132; and Ebstein, "D. Arch. f. klin. Med.," vol. LVIII, p. 14.

with certainty, it may be accepted as a possible complication through the trifacial nerve, in view of the frequency of other neuralgic manifestations in influenza.

7. PAROTITIS EPIDEMICA (MUMPS).

In this obscure epidemic disease, which belongs to the class of infectious diseases, the general infection manifests itself in various parts of the body, showing that the typical swelling of the parotid gland is only a local expression of the general disease. The commonest complication—that of orchitis and epididymitis—is as little understood as the occasional involvement of the ear.

The aural complication usually takes the form of labyrinthine deafness, appearing, as a rule, during the first days of the disease, along with other symptoms of Ménière's complex, and offering an obstinate resistance to every mode of treatment, while the accompanying symptoms of vertigo, tinnitus aurium, and disturbances of the equilibrium subside. Like the complications in the sexual organs, those in the ear show a predilection for the age of puberty, being most frequent between the tenth and twentieth years.[1] The total number of cases reported is very small. In 1884 Connor was able to collect 34 cases, and in 1883 Gradenigo could report only 38 positive observations of deafness due to mumps. One or both ears may be affected, and there appears to be no connection with the situation of the primary disease if the latter has been unilateral. There have even been reported rudimentary cases in which orchitis and deafness were present without glandular swelling (Gradenigo's case). As the prognosis is absolutely unfavorable, the disease may, if it be bilateral and occurs in early infancy, lead to deafmutism, the frequency of which is given as 0.3% by Mygind, in the Saxon deaf and dumb statistics, and as 0.5% by American statisticians.

The otoscopic picture is in every respect negative, and there is absolutely no proof that inflammations of the tympanic membrane and exudations in the middle ear have anything to do with the disease. Functional test shows deafness or marked reduction in the hearing of the internal

[1] Gradenigo, "Schwartze's Handb.," II, p. 440.

ear, while, according to Moos,[1] the power of hearing for the lower notes and bone conduction may be preserved.

Numerous attempts have been made to explain the deafness of infectious parotitis, but they are all more or less improbable, and therefore of no interest.

The subject will be found discussed at length in papers by Rossa,[2] Moos,[3] Haug,[4] Gradenigo,[5] and Alt.[6]

Pilatti [7] describes a case of parotitis in which tracheotomy was required on account of edema of the larynx.

8. ACUTE RHEUMATOID ARTHRITIS (POLYARTHRITIS RHEUMATICA ACUTA).

One of the first diseases in which the tonsils were recognized as the port of entry for a general infection was acute articular rheumatism. The importance of angina in the etiology of this disease was first pointed out by Lagranère, Boeck, Loebl, Mantle, and others, all basing their assertions on clinical observations.

But the confusion that still prevails with regard to the cause of acute articular rheumatism was not removed by the bacteriologic examination of cases of rheumatoid angina, for the greatest variety of microorganisms—staphylococcus aureus, pyogenic streptococci, streptococcus citreus, and pneumococci—was found. As this is not the place for a detailed theoretic discussion of the relation between the angina and rheumatism,—which will be found, together with a complete report of all the cases in the literature, in the works of Buss,[8] Suchannek,[9] and Bloch,[10]—I shall merely refer briefly to the clinical observations that have been reported. Any one of the varieties of tonsillitis, both catarrhal and follicular, may appear either as a forerunner of rheumatism before the joints are affected, or as a feature of the fully developed clinical picture. The complication can not at the present time

[1] "Berlin. klin. Wochen.," 1884, No. 3.
[2] "Zeitschr. f. Ohr.," vol. XII. [3] "Schwartze's Handb.," I, p. 584.
[4] "Die Krankh. des Ohres," etc., p. 75.
[5] "Schwartze's Handb.," II, p. 439.
[6] "Mon. f. Ohr.," 1896, p. 525.
[7] See "Semon's Centralbl.," VIII, p. 149.
[8] "D. Arch. f. klin. Med.," vol. LIV.
[9] Bresgen's Sammlung, vol. I, H. 1.
[10] "Münch. med. Wochen.," 1898, Nos. 15, 16.

be regarded as a rare occurrence in Germany, as stated by Wagner [1] in 1878, and its frequency shows that it is not an accidental coincidence, but that it represents a symptom of the general disease. Gerhardt [2] mentions, as a strong proof of internal connection between tonsillitis and rheumatism, a case of Staffel's,[3] in which an attack of articular rheumatism rapidly followed a severe inflammation of the tonsils, and the articular affection was removed only after methodical treatment of the mouth. In addition to tonsillitis and pharyngitis, we also have catarrhal laryngitis; but by far the most important diseases of the larynx, from a practical point of view, are those which must be regarded as typical manifestations of the rheumatic infection. They may be divided into two varieties, which have been designated respectively as disease of the joints of the larynx and as laryngitis acuta rheumatica circumscripta (Nodosa).

The crico-arytenoid articulation is the only one that has been known to be involved in an acute articular rheumatism, although the fact that there is no report of the crico-thyroid joint being involved may be due to defective diagnosis, and it seems to me that Meyer's [4] case might easily be regarded as one of this kind, since the laryngoscopic findings were negative. Rheumatism of the crico-arytenoid articulation is usually bilateral, and manifests itself in the laryngeal image in redness and swelling of the arytenoid region and in sluggishness or arrest of the vocal cords, simulating paralysis. Besides the aphonia, the subjective symptoms consist in a sensation as of a foreign body, dyspnea, and dysphagia, all of which, according to Meyer's description, are worse when the patient lies down. An important diagnostic point is the tenderness over the crico-arytenoid joint or over the thyroid cartilage; in the latter case the symptom possibly points to disease of the cricothyroid articulation. The laryngeal complication usually develops between the fourth and the twelfth day after the onset of the articular rheumatism; the prognosis is favorable, recovery usually occurring in a short time (according to Meyer, in a week). Grünwald [5] mentions a case of "cadaver position"

[1] Wagner, "Ziemssen's Handb.," VII, p. 148.
[2] "Verhdl. des Congr. f. inn. Med.," 1896, p. 180.
[3] "Zeitschr. f. prakt. Aerzte," 1896, No. 4.
[4] "Berlin. klin. Wochen.," 1894, No. 16.
[5] "Berlin. klin. Wochen.," 1892, No. 20.

on the right side after articular rheumatism which was cured in two years. The disease usually responds promptly to salicylic acid. The reported cases, which are very few in number, have been collected by Lacoarret [1] and Sendziak [2]; Archambault's [3] thesis is also well worth reading.

The second form of rheumatic disease in the larynx is described by Uchermann [4] as laryngitis acuta rheumatica circumscripta (nodosa), although Goldscheider [5] lays claim to priority, as he reported an analogous case in an earlier paper. The condition occasionally manifests itself in connection with erythema nodosum as a "circumscribed reddish or bluish-red, moderately firm infiltration, very sensitive to the touch," which may attain a considerable size (as large as an almond), seated usually in the neighborhood of the crico-arytenoid articulation or in the aryepiglottic fold; in the former situation pseudo-ankylosis, with immobility of the vocal cord, is likely to result.

As the inflammation also invades neighboring portions of the larynx, and thus leads to edema both in the aryepiglottic folds and on the epiglottis, the symptoms of dyspnea and dysphagia may be added.

The prognosis in this form also is favorable.

Wolf [6] described two cases of acute inflammation of the middle ear in acute articular rheumatism. In the first case both ears were affected one after the other; one of the ear-drums ruptured spontaneously; in the other, paracentesis was required. The rheumatism was very severe, and did not appear in the joints until several days later; the suppuration which followed the inflammation was cured in four weeks. In the second case the aural disease was followed after only nine days by diffuse swellings in the joints. From the fact that in both cases the impairment of hearing and thickening of the ear-drum were permanent, Wolf concludes that articular rheumatism may be the cause of sclerotic catarrh in the middle ear. A similar case is reported by Ménière. [7] We have no knowledge of disease in the joints of the ear ossicles; the possibility of rheumatism in the joint between the malleus and the incus, and between the incus and the stapedius, is, however, worth considering.

[1] "Rev. de lar., d'ot. et de rhin.," 1891, No. 11.
[2] "Arch. f. Laryng.," IV, p. 264, and VI, p. 168.
[3] Thèse de Paris, 1886.
[4] "Deutsche med. Wochen.," 1897, p. 749.
[5] *Ibid.*, p. 807.
[6] "Arch. f. Ohr.," 41, p. 213.
[7] "Rev. mens. de lar., d'ot., et de rhin."

The cases reported by Bloch [1] in which disease of the ear is said to have produced an acute articular rheumatism do not seem to me sufficiently convincing to justify the assumption of a new mode of infection for that disease.

9. DIPHTHERIA.

The description of diphtheria belongs to the domain of internal medicine,[2] and the manifestations of the disease in the nose, pharynx, and larynx will be found amply discussed in the text-books.

I shall not, therefore, attempt to give a description, as it does not belong to the scope of this work, and would, if it made any pretensions to thoroughness, occupy too much space. Instead, I shall confine myself to a discussion of the sequels occurring after diphtheria in the nose, pharynx, and larynx, and in the ears.

In the pharynx and larynx we have post-diphtheric palsies of both the sensory and motor nerves, the cause of which is now generally conceded to be a peripheral neuritis. The time of their appearance is usually given as from two to six weeks after the diphtheria. The paralysis affects most frequently the uvula. The nature of the paralysis is unmistakable, as it can be seen by direct inspection, and manifests itself, besides, in the conspicuous symptoms of dysphagia, regurgitation of liquids through the nose, and nasal speech. Although this form of paralysis has occasionally been observed early, following immediately upon the pharyngeal disease, it must be remembered that a paretic condition of the palatal muscles may be produced by the diphtheric disease of the mucous membrane invading the deeper-lying muscles.

Anesthesia of the pharyngeal and laryngeal mucous membrane is much more rare. It was observed in the cases cited by v. Ziemssen [3] and elsewhere.

Paralysis of the vocal cords has been observed with at least sufficient frequency to remove any doubt of its occurrence, and it is difficult to understand what could have led Baginsky [4] to say that "he was unable to find among

[1] "Münch. med. Wochen.," 1898, Nos. 15, 16.

[2] The latest description is by Baginsky, in Nothnagel's "Spec. Path. u. Ther.," II. Bd., I. Th.

[3] In "v. Ziemssen's Handb.," vol. IV, p. 405.

[4] *Loc. cit.*, p. 215.

all the reported cases any description of paralysis of the crico-arytenoidei postici due to lesion of the recurrent laryngeal nerves after diphtheria; he himself had certainly never seen it." Von Ziemssen [1] reports two cases of diphtheric paralysis of the pharynx, larynx, and extremities. In one case the left vocal cord was completely paralyzed in the cadaver position, while the right was very sluggish and limited in its excursions.

I once saw a doubtful case in which a unilateral complete paralysis of the uvula and vocal cords was associated with anesthesia of the mucous membrane and abolition of all the reflexes. The paralysis occurred about six weeks after a mild case of diphtheria, and after it had lasted seven weeks the vocal cord gradually returned to the median position and finally completely regained its movability.

Clifford-Beacher [2] observed a case in which paralysis of the adductors followed that of the abductors, while recovery took place in the inverse order.

According to Lublinsky, [3] postdiphtheric paralysis of the vocal cords is more frequent, and occurs earlier when the serum treatment is employed; in one case he saw it as early as the ninth day of the disease.

The prognosis of postdiphtheric paralysis is favorable. In anesthesia and impairment of the reflexes in the upper air-passages there is some danger of inspiration pneumonia.

In addition to these peripheral palsies there have been observed paralyses of central origin, probably due to hemorrhage, manifesting themselves under the form of hemiplegia and presenting the symptoms of paralysis of the uvula and aphasia. It is not stated whether or not the vocal cords were also paralyzed. Edgren [4] gives a review of the cases reported in the literature, adding some of his own.

Diseases of the ear in diphtheria may be divided into—

1. Diphtheric inflammations of the external auditory meatus.

2. Diphtheric inflammations of the tube and of the middle ear.

3. Acute catarrhal and purulent inflammations of the middle ear without the formation of membranes.

[1] *Loc. cit.*, p. 215. [2] "Semon's Centralbl.," IX, p. 86.
[3] "Deutsche med. Wochen.," 1895, No. 26.
[4] "Deutsche med. Wochen.," 1893, No. 36.

1. Diphtheria of the external auditory meatus is very rarely seen. The only reliable observation of its occurrence in connection with pharyngeal diphtheria is that of Treitel, [1] while in the other published cases of croupous inflammation of the external meatus, by Wreden, Blau, and others, the diagnosis of true diphtheria is not positive, some of the cases representing the scarlatinal variety. In Treitel's case, diphtheric membranes were found in both ears, representing a complete cast of the external auditory meatus. The inflammatory symptoms were very marked, and there was extensive swelling over the mastoid process. The disease extended to the auricle, but the ear-drum remained intact.

A bacteriologic examination was made by Kossel, and was negative, although he found rod-shaped organisms resembling the diphtheria bacillus; Treitel attributes the negative outcome of the cultures to the sublimate solution in which he had preserved the membranes before they were examined.

2. As regards diphtheric disease in the tube and in the middle ear, we do not possess any positive investigations supported by the bacteriologic demonstration of diphtheria bacilli, but we are forced by the result of autopsies and by clinical observation to assume the occurrence of such complications in true diphtheria.

When we attempt to analyze the reported cases, most of which belong to the prebacteriologic period, or else are so little to be relied upon as to be quite unworthy of discussion, it is often difficult to separate cases of false from those of true diphtheria. Wreden [2] and Burkhardt-Merian, [3] for instance, discuss scarlatinal diphtheria and true diphtheria and their complications with croupous inflammation of the middle ear without making any distinction between them. On the other hand, we find in the observations of Wendt, [4] Küpper, [5] Moos, and Hirsch [6] the necessary materials for a description of diphtheric disease of the ear.

The middle ear may be affected alone or in combination

[1] "Deutsche med. Wochen.," 1893, p. 1388.
[2] "Mon. f. Ohr.," vol. II, p. 148.
[3] Volkmann's "Sammlung klin. Vortr.," I. Reihe, Serie VII, No. 182.
[4] "Arch. f. Heilkunde," XI and XIII.
[5] "Arch. f. Ohr.," XI, p. 20.
[6] "Zeitschr. f. Ohr.," XIX, p. 101.

with the tube. Diphtheric membranes are found adhering to the mucous membrane of the tympanic cavity or covering the ossicles or lining the cells in the bone. In a case of acute purulent otitis media after diphtheria, reported by Lommel,[1] beginning membrane formation was found in individual mastoid cells.

The symptoms of the disease are those of any acute otitis media, rise of temperature and pain being the most prominent; the pain is aggravated by the fact that the ear-drum shows no tendency to spontaneous perforation, so that expulsion of the membranes into the external meatus occurs only after paracentesis has been performed.

The course of a croupous disease of the ear following diphtheria appears to be the same as that of one following scarlet fever; both diseases are considered equally malignant as regards destruction of the walls and of the ossicles in the middle ear, the production of extensive caries in the temporal bone, and extension to the labyrinth, so that the prognosis must be regarded as unfavorable.

Nothing definite is known in regard to the frequency of true diphtheria in the ear. It is certainly very rare, and does not bear any proportion to the frequency of scarlatinal diphtheria.

3. It has been demonstrated by anatomic investigations—among which those of Wendt and Lommel [2] are worthy of special mention—that even without clinical appearances, and certainly without any involvement of the drum membrane, certain alterations are regularly found in the middle ear of diphtheric cadavers which we must regard as due to catarrhal otitis media with or without serous exudation, catarrhal otitis media without purulent but with mucous secretion, or acute purulent otitis media. Although Lommel found pus in the middle ear in one-half of his cases, the ear-drum was never perforated nor even markedly congested, showing that a clinical diagnosis based on the appearance of the otoscopic image would have been impossible.

This explains why the anatomic findings of Lommel in regard to the frequency of aural complication in diphtheria are in direct opposition to clinical observations. While, on

[1] "Zeitschr. f. Ohr.," XXIX, cases VII and XXIV, p. 301.
[2] "Zeitschr. f. Ohr.," XXIX, p. 301.

the one hand, Lommel found the ear intact in only 1 out of 25 autopsies of diphtheric cadavers, and therefore laid down the rule that otitis media forms an integral part of the clinical picture of "diphtheric disease of the respiratory organs," Baginsky,[1] on the other hand, reports that although he examined the ears of his diphtheria patients with the greatest care, he found only from 5% to 6% in which an inflammation was present. Hence we must not overestimate the significance of these findings from a clinical point of view, and as in my cases the reports show that the alterations in the mucous membrane of the middle ear were very slight and analogous to those which are found in other infectious diseases,—especially measles (Rudolf and Bezold),—we must assume that they undergo regeneration without giving rise to any clinical symptoms.

As has been stated in connection with croupous inflammation of the middle ear, the tube may remain intact. Lommel found that the cartilaginous extremity was rarely attacked, while the "main central portion" was regularly free from any inflammatory process, even in one case where there was a diphtheric exudate about the orifice itself. Hence, direct extension of the inflammation from the pharynx to the middle ear is to be regarded as unusual, the middle-ear disease being rather the expression of the general infection; and I may remark that, in harmony with this statement, consecutive ear disease after nondiphtheric tonsillitis, whether of the catarrhal, lacunar, or suppurative variety, is rare, notwithstanding the fact that those diseases are usually referred to in the text-books as frequent etiologic factors in suppuration of the middle ear.

Lastly, it appears that nerve deafness may occur after diphtheria; it is probably due to toxic influences, and belongs to the class of postdiphtheric palsies. The cases reported are so few [2] [3] and so incomplete that it is impossible to draw any conclusions from them.

[1] "Diphtherie und diphtheritischer Croup," in Nothnagel's "Spec. Path. u. Ther.," Bd. II, 1. Th., p. 258.

[2] Kretschmann, "Arch. f. Ohr.," XXIII, p. 236.

[3] Haug, "Die Krankh. des Ohres," etc., p. 69.

10. ERYSIPELAS.

Primary erysipelas of the mucous membrane of the upper air-passages is a very rare occurrence, and its pathology and clinical course can not readily be distinguished from those of other infectious diseases of the mucous membrane attended with high fever, redness, swelling, edema, and leading finally to abscess formation. Indeed, various authors have objected to applying the term erysipelas to any disease of the pharynx or larynx. Kuttner [1] and Semon [2] are probably quite right in advocating the adoption of the general term "acute septic inflammations of the larynx," rejecting the terms erysipelas of the pharynx and larynx, phlegmon, angina (Ludovici), or acute edema of the pharynx and larynx as being merely synonymous terms for the same clinical picture. Cases of undoubted erysipelatous infection of the mucous membranes of the throat, while rare, are none the less of the highest importance, as primary erysipelas of the mucous membrane of the nose, pharynx, larynx, and mouth may, by extension to the external skin, give rise to secondary facial erysipelas. This once occurred in Schwartze's [3] ear clinic: a patient who had had a pharyngeal tonsil removed went to see an erysipelatous patient and contracted erysipelas of the nasopharynx, which spread through the tubes to the middle ear, and from there to the external meatus, the auricle, and the face. Rendu [4] saw a case of erysipelas, where the diagnosis was confirmed by bacteriologic examination, in a man suffering with syphilitic glossitis; there was a fresh rise in the temperature when the erysipelas spread to the face. Garel [5] describes a case of erysipelas which began in the tongue and reached the face by way of the pharynx and nose.

Erysipelas occasionally occurs as a remote consequence of disease of the anterior nares, of the auricle, and of the external auditory meatus, for excoriations and rhagades due to chronic eczema may form the port of entry for the germs of the disease. That this is the mode of infection is proved by the subsequent extension of the erysipelas, which in

[1] "Larynxödem und submuköse Laryngitis," Berlin, 1895, Georg Reimer.
[2] "Med. chirurg. Transactions," vol. LXXVIII, 1895.
[3] "Arch. f. Ohr.," vol. XXXVIII, p. 213.
[4] "France méd.," 1892; see "Semon's Centralbl.," X, p. 131.
[5] "Ann. des malad. de l'oreille," etc., 1891, No. 5.

such cases first appears in the neighborhood of the nose and ear, and gradually extends from those points to the skin of the face and head. This variety often shows a tendency to recurrence, and habitual facial erysipelas [1] is usually due to chronic eczema of the nose or of the ear.

This etiologic sequence is important from a therapeutic point of view, as the occurrence of erysipelas can be guarded against only by combating the eczema and the basal disease which is responsible for the eczema, such as chronic rhinitis or suppuration from a neighboring cavity or from the ear.

A suppuration from the middle ear due to erysipelas, like any other suppuration, may extend to the labyrinth and produce symptoms in that locality, as shown in a case reported by Schwartze. I can not imagine what Haug [2] means when he says that "the internal ear itself probably escapes, in some cases at least, in so far as the inflammation does not extend to the labyrinth; at most there may be signs of a temporary congestion," nor am I much impressed by the elegant phrase that "erysipelas not rarely reaches its terminal phase in the periauricular lymphatic glands."

11. MALARIA.

We find numerous statements in regard to the occurrence of vasomotor rhinitis and hydrorrhœa nasalis in malaria. Chapell [3] has collected a series of cases in which the hydrorrhea occurred periodically, corresponding to the malarial attacks.

Whether epistaxis is to be regarded as a characteristic symptom of the disease or not, is still a matter of doubt.

According to Löri,[4] we rarely have in malaria the "typical occurrence of aphonia." On various occasions he observed hoarseness or aphonia, synchronous with the attack, occurring as early as the algid stage and disappearing as the temperature fell. In these "intermittent aphonias" he always found, "on laryngoscopic examination, paralysis of all the muscles supplied by the recur-

[1] Comp. Friedrich, "Pachydermie im Anschluss an habituelles Gesichtserysipel," "Münch. med. Wochen.," 1897, No. 2.

[2] "Die Krankh. des Ohres," etc., 1893, p. 107.

[3] See "Semon's Centralbl.," XI, pp. 395 and 508.

[4] "Die Veränderungen des Rachens," etc., p. 156.

rens—sometimes only on one side, sometimes on both." Edema of the larynx, according to him, is an occasional symptom of the malarial cachexia.

Haug [1] has given us a comprehensive presentation of malarial diseases of the ear in which the literature is fully quoted. Protozoic origin has been assumed for certain diseases of the ear which occur in periodic attacks, corresponding to the type of malaria, at intervals of from one to three days, and present the picture of an acute inflammation of the middle ear or of nervous deafness without being necessarily accompanied by other malarial symptoms. Even the older physicians were well aware of the fact that intermittent otalgia sometimes occurred in the course of intermittent fever, and Schoenlein [2] states that the neuralgia may be localized in the posterior auricular nerve and in the chorda tympani, which, as Voltolini adds in explanation, "shows that the pain is felt in the interior of the ear, as the chorda tympani itself is not capable of giving rise to neuralgia."

As Weber-Liel [3] was the first to point out the connection between "otitis intermittens" with malaria, and gave clinical histories in support of his assertion, I shall quote his description of the form of malaria which is attended with acute irritation of the ear : "After an attack of tonsillitis and catarrh of the nasopharynx, at least in most cases, the aural affection usually appears toward evening or during the night, accompanied by chills, which may be more or less marked or only barely perceptible. At first there is only an uncomfortable sense of fullness and buzzing in the ears, while not rarely a feeling of pressure in the head and vertigo are among the first symptoms. The patient passes a restless night, perspires profusely, but feels quite well on the following day." These phenomena recurred after the manner of malaria for two or three days ; the ear-drum and the external meatus were very hyperemic ; the middle ear was the seat of a serous or serosanguineous exudate corresponding in quantity to the frequency of the attacks, and in some cases perforation of the ear-drum occurred, followed by serosanguineous or purulent discharge, as was also observed by Haug.[4] For an explanation of this symp-

[1] "Die Krankh. des Ohres," etc., p. 145.
[2] Quoted by Voltolini, "Mon. f. Ohr.," 1878, p. 57.
[3] "Mon. f. Ohr.," 1871, p. 125.
[4] "Mon. f. Ohr.," 1878, p. 59.

tom-complex we are driven to assume a trophoneurosis of the trifacial nerve.

Of the second form of malarial disease Garzia [1] gives the following description, based on the observation of 24 cases: After a rise of temperature, pain and deafness appear in both ears, the pain disappearing as the fever subsides, while the deafness remains. According to Haug, all kinds of subjective noises may make their appearance periodically.

The diagnosis for both forms of the aural disease is based on the intermittent type, the exposure of the patient to malarial infection, and the beneficial effects of quinin, which are said to be very striking and even capable of curing the deafness of the second form.

[1] "Verhandl. des internat. Congresses in Rom," reported in "Arch. f. Ohr.," XXXVII, p. 258.

VII. CHRONIC INFECTIOUS DISEASES.

1. TUBERCULOSIS AND LUPUS.

Tuberculosis manifests itself in all its various forms in the upper air-passages. The anatomic process is analogous to that seen in all mucous membranes, presenting as its chief type that of tuberculous infiltration, with tubercle formation in the submucosa and mucosa, followed by ulceration and granulation. It will be shown in a later chapter how these fundamental types can readily be classified by their clinical appearances into separate subdivisions, which tend to make the picture of tuberculosis appear somewhat more complicated than it really is when its mode of origin is thoroughly understood. But before going into that question we must adopt some theory as to how tuberculosis originates in the upper air-passages. The mode of infection has given rise to much discussion, and various opinions have been advanced in regard to the path by which the tubercle bacillus, the causative agent in all the various forms, effects an entrance into the tissues. The mode of origin depends largely on whether the tuberculosis is considered as a primary or as a secondary disease, since if the pathogenic germs first become localized in the upper air-passages, the infection may be derived from the inspired air and the food ingested; while if we assume a primary tubercular focus in other organs,—as, for instance, the lungs,—secondary infection of the upper air-passages may take place either from within, by way of the lymphatic and vascular channels, or from without, by direct infection of the mucosa through the agency of tubercular sputa. The first of these two groups—that of primary tuberculosis of the throat, nose, and larynx—is comparatively rare. It is only recently that it has achieved general recognition, and in the case of the larynx, its existence is still a matter of dispute. The most recent studies in the mode of tubercular infection have led to the careful investigation of the various lymphatic elements in their relation to tuberculosis.

Though formerly primary tuberculosis of the palatal, lingual, and pharyngeal tonsils was not believed to occur, the present tendency, since Strassmann's [1] investigations,—in the course of which he found tonsillar tuberculosis in 13 out of 21 tuberculous cadavers,—is to regard not only tuberculosis in general, but also primary infection of the pharyngeal lymphatic ring as of comparatively frequent occurrence. Clinical observation has not been able to keep pace with anatomic investigation on account of the difficulty of diagnosing latent tonsillar tuberculosis with any degree of certainty. The palatal and pharyngeal tonsils show no macroscopic alterations in cases in which they appear manifestly tuberculous under the microscope; as a rule, they were found to be only slightly hypertrophied, while in a somewhat larger proportion of cases small atrophic and brawny nodules were observed. As far as I know, Ruge's [2] case, in which the clinical diagnosis of latent tonsillar tuberculosis was confirmed by subsequent examination of the extirpated tonsils, is the only one of its kind, and even in this case the symptoms were very vague. The patient, a girl eighteen years old, had had "enlarged tonsils" since childhood, and for some time had complained of a vague feeling of discomfort in the throat; later, Pott's disease of the cervical vertebræ developed. A few cases [3] have been reported in which extirpation of the pharyngeal or palatal tonsils was followed by a fatal pulmonary tuberculosis within a period of from one to two years, probably as the result of a recrudescence of a latent tonsillar tuberculosis and the effect of surgical interference. Tuberculosis of the lymphatic structures of the pharynx is usually attributed to direct bacillary infection by the respiratory air current or the food; and it is not necessary in either of these modes of infection to suppose an abrasion of the epithelium which should afford a port of entry to the pathogenic germs, for Stöhr, [4] Suchaneck, [5] and Lexer [6] have been able to demonstrate the possibility of the germs gaining entrance through sound mucous membrane of the pharyngeal structures. This affords a strong argument for the possibility of a latent tonsillar tuberculosis giving rise to a descending

[1] "Virch. Arch.," XCVI, p. 319. [2] "Virch. Arch.," CXLIV.
[3] Kafemann, "Bresgen's Samml.," II, H. 4–5.
[4] "Virch. Arch.," XCVII.
[5] "Ziegler's Beiträge," 1888. [6] "Arch. f. klin. Chir.," Bd. LIV.

tuberculous infection of the cervical lymphatic glands. The relation of such cases to those in which there is a coexistent tuberculosis of the lungs and larynx—that is to say, whether they represent a primary infection which has become latent, or one secondary to the pulmonary and laryngeal affection—can not at present be determined with certainty. A few authors maintain the possibility of primary tuberculosis of the pharynx, but its occurrence is at least doubtful.

The study of tuberculosis of the nose has established the possibility of primary tuberculosis in this organ. This statement is based not only on clinical investigations,—in many cases all the other organs were found to be free from tuberculosis,—but also on the favorable effect of removing the tubercular tumors which are often found on the cartilaginous septum [1] of the nose. It is evident that in this form of nasal tuberculosis we have to deal with a primary infection. This region of the septum plays an important part in the pathology of the nose, as it is the point where the inspiratory air current first impinges on the septum after passing through the vestibule, and deposits any foreign body which it may contain. In this way erosions on the septum occur which lead to the sequel known as xanthosis, and it is at this point, where the nutrition is normally low, that the tubercle bacillus is apt to establish itself in favorable subjects and to lead to tubercular ulceration or tumor formation.

Primary tuberculosis of the larynx must be regarded as exceptional; indeed, we should be inclined to deny its occurrence altogether were it not for the positive postmortem proof afforded by the two examples of Orth and Demme and the statement of M. Schmidt, based on a large experience, that "it is particularly apt to occur in the form of tumors on the vocal cords and ventricular bands," although M. Schmidt himself points out the lack of postmortem evidence. The theory of primary tuberculosis of the larynx, which is doubted even by Störk and Schrötter, finds little, if any, confirmation in Aronsohn's paper,[2] as his cases are not above criticism, and an analysis of cases published elsewhere yields only three instances where the lungs were

[1] Chiari, "Arch. f. Laryng.," I. Koschier, "Wien. klin. Wochen.," 36, 37, 39, 40–42, 1895.
[2] "Arch. f. Laryng.," V.

found intact at the autopsy. We can not admit as proof of primary laryngeal disease cases in which the lungs are found to be affected at the autopsy, even when we find the assertion that the lung disease is of more recent origin than the laryngeal affection. Primary tubercular chondritis and perichondritis may possibly occur; cases of perichondrial tubercular abscess on the exterior surfaces of the thyroid cartilage, unaccompanied by other laryngeal or pulmonary manifestations, are occasionally met with, and, as I have had occasion to observe, such cases, if operated on, yield a favorable prognosis. Angelot [1] and Catti [2] have described cases of acute miliary tuberculosis beginning in the pharynx and larynx. Angelot's case terminated fatally in from two to six months; the two cases by Catti on the eighth and ninth day, respectively. The latter author emphasizes the fact that the laryngeal symptoms may be so prominent as to mask any morbid symptoms in other organs and to suggest diphtheria.

The most frequent, not to say regular, form of infection met with in the upper air-passages is the secondary one; but here again opinions diverge as to whether the infection is brought about by direct contact with the infected sputum or through the lymphatics and blood-vessels. The former opinion may be called that of the morbid anatomists, as we find among its representatives such names as Orth [3] and E. Fränkel,[4] while the other is held chiefly by laryngologists, such as Korkunoff (v. Ziemssen's clinic),[5] Schnitzler, Schrötter, and others; but it is worthy of remark that neither of the two factions considers its own view as the only possible explanation, and admits the possibility of the opposite mode of infection in isolated cases. Orth says: "When we have to deal with a typical case, where, perhaps, there is only a large ulcerated cavity in one apex; where all the bronchi through which the secretions from this cavity must pass during expectoration are full of tubercular ulcers; where we find smaller ulcers only on that side of the main bronchus and lower portion of the trachea which, from the position of the body, must come into contact with the secretion, and the ulcers are found

[1] Quoted by Orth, p. 323. [2] "Wien. klin. Wochen.," 1894, p. 438.
[3] "Lehrb. der spec. path. Anat.," p. 320.
[4] "Virch. Arch.," CXXI, p. 523.
[5] "D. Arch. f. klin. Med.," XLV, p. 43.

to increase in size and frequency as we ascend; where, omitting a part of the trachea, the tubercular affection is seen to be more extensive wherever the walls of the air-passages are approximated, and the sputum is therefore forced against the sides,—the conclusion seems inevitable that the sputum constitutes the vehicle by which the tubercular toxin is conveyed from the cavity and deposited during its transit through the air-passages on favorable regions of the mucous membrane." Such "inoculation" is, of course, quite conceivable, and the formation of ulcers by the entrance of bacilli from the exterior, either through excoriations or through the intact epithelium, is possible; but, instead of regarding it, with E. Fränkel, "as the essential and primary mode of infection," would it not be more logical to view it only as an occasional factor in the etiology of the disease?

The strongest argument in the hands of those who believe that the infection takes place through the vascular and lymphatic channels is found in the morbid anatomy of laryngeal tuberculosis. The first stage of the disease is characterized by the deposition of tubercles within the mucosa at a greater or less distance from the epithelium, which at first retains its integrity; in fact, there is frequently a broad, wide zone of healthy tissue between the infiltration and the epithelium. In the laryngoscopic image tubercular infiltration of this kind, which may become quite extensive through the subsequent formation of a large number of tubercles, manifests itself in a circumscribed swelling covered with healthy mucous membrane. These conditions can be studied in preparations of tubercular larynges, and thus we have a confirmation of the excellent descriptions given at first by Heinze,[1] and more recently by Korkunoff[2] and others. It is only later, when the tubercle increases in size and reaches the level of the epithelium, that the latter begins to degenerate; the membrane becomes loosened and the epithelium breaks down into detritus. In this way a tubercular ulcer is formed, the superficial necrosis keeping pace with the progress of the tubercular infiltration. The distribution of the tubercle bacilli also corresponds with these anatomic conditions. Korkunoff found that while the outer layers of the epithe-

[1] "Kehlkopfschwindsucht," Leipzig, Veit & Co., 1879.
[2] "D. Arch. f. klin. Med.," vol. XLV, p. 43.

lium contained few bacilli, the deeper portions, nearer the tubercles, contained large numbers. The anatomic conditions, therefore, would appear to show that the tubercular process spreads by way of the lymphatic or vascular channels, and this is in accord with daily clinical experience, for we frequently find that apparently harmless thickenings, especially on the posterior wall, often undergo a bluish discoloration, become necrotic, and are converted into ulcers, so that it does not seem plausible in these cases of tubercular infiltration to suppose an infection by contact notwithstanding that Orth refuses to admit the explanation of the subepithelial appearance of the tubercles. There is no doubt that tubercles produced by contact do occur in the epithelium of the larynx, but they are of an entirely different nature, both anatomically and clinically. They were formerly described as diphtheric (Rokitansky), then as aphthous erosion and corrosion ulcers ; a difference of opinion concerning their origin existed for a long time, as it seemed doubtful whether they should be explained as simple tubercular or merely as arrosion ulcers, due either to irritation of the mucous membrane by the contents of the cavity or to a secondary infection of superficial erosions.

These ulcers are not the result of an infiltration, as was formerly believed, but represent superficial miliary tubercle nodules in process of degeneration. They form flat superficial ulcerations with a decided tendency to spread, while the tendency to form granulations in the floor of the ulcer, which is such a marked clinical characteristic of other tubercular lesions, is absent. The floor of the ulcer is covered by a thick, yellowish exudate, which sometimes forms a true fibrinous membrane slightly raised above the level of the surrounding parts. It is probably this appearance that induced Rokitansky to describe them as diphtheric ulcers.

These arrosion ulcers represent, therefore, another specific expression of the tubercular process, ultimately due to the action of the tubercle bacilli, but their mode of infection is evidently quite different from that which I have so far described. Since from the very beginning of the disease the tubercular infiltration is superficial, we can not in this case suppose a movement of the bacilli from within outward, —in other words, from the vascular or lymphatic channels,

—and must admit the explanation of an infection by contact with tubercular sputum. The significance of a mixed infection with staphylococci and streptococci has not as yet been determined, but such an infection appears probable when we consider the rapid spread of these ulcers.

Certain clinical arguments have been advanced to prove the occurrence of infection from the lungs and larynx through the blood-vessels, but the observations of Friedreich, Schrötter, and Schech (which were not confirmed by Heinze's postmortem investigations), that the disease always affects the organs on the same side, are not above criticism. If we accept direct infection of the larynx as the rule, it is at least remarkable that when the expectoration is very copious and contains bacilli, there is no laryngeal disease; whereas it is present in cases when there is little or no sputum in a beginning pulmonary tuberculosis, and there is therefore no possibility of long-continued contact of the sputum with the mucous membrane, favoring the entrance of the bacilli. To meet this objection, Orth assumes a certain constitutional predisposition or weakness of the mucous membranes to explain the occurrence of infection by contact. But if contact with the sputum plays such an important rôle in predisposed individuals, why does the disease become localized in the larynx? Does not the squamous epithelium in the deeper portion of the pharynx, in the pyriform sinuses, and on the posterior and lateral pharyngeal walls present the same possibility of infection from without as the epithelium of the larynx, which shows a special preference for tubercular disease in those portions covered by squamous epithelium? The sputum collects in much larger quantities in these regions than it does in the larynx itself, where it is constantly expelled by reflex cough, and therefore infection by contact would be quite as likely to occur as in the larynx; but, as a matter of fact, this is not the case. We know from the observation of other laryngeal diseases, especially carcinoma, that enlargement of the lymphatic glands and extension to the surrounding structures occur only in the later stages of the disease, and it appears that the lymphatic system of the larynx occupies, in a certain sense, a unique position. Of course, we can not as yet say with any certainty that this factor in any way contributes to the tendency of the infection to localize itself in the larynx, to the exclusion of other por-

tions of the upper passages, but the observation is worthy of consideration.

We therefore reach the conclusion that both views in regard to secondary infection of the larynx from the lungs have their pros and cons, and that it is impossible to draw any absolute theoretic deductions in support of either theory. In view of our clinical and anatomic experience, we recognize infection of the larynx by way of the lymph-channel, as probably more frequent, and reserve infection by contact for those cases which manifest themselves in the form of arrosion ulcers.

Tuberculosis of the nose manifests itself in three different forms :

(*a*) Tuberculoma.

(*b*) Extensive infiltration with ulceration.

(*c*) Bone disease with secondary extension to the mucous membrane.

The typical seat of tuberculous tumors is the cartilaginous septum, although in a few cases they are found on the bony portion. They appear as tumors with a broad base, imperfectly circumscribed, and of varying size, so that they lead to a greater or less constriction of the nasal cavity. The epithelium is usually preserved and appears healthy on the surface ; the mucous covering is smooth ; the surface is either uniform or slightly bosselated ; occasionally, several distinct nodules can be made out on the tumor. They show very little tendency to ulceration and caseation of the contained tubercle ; it is only in very old cases that there is occasionally seen a tendency to ulceration at the apex of the tumors (Koschier). The swelling usually appears first on one side of the septum, but later a similar swelling is seen on the opposite side, so that we have two dark-red or grayish-red tumors, which can be seen without the aid of a reflector and resemble traumatic abscesses of the septum.

In this stage of the disease the perichondrium becomes the seat of round-celled infiltration ; the process invades the cartilage of the septum, which undergoes necrosis ; the dividing wall between the two tumors breaks down, and they become fused. This destructive process may go on for years without any marked alteration in the clinical picture. In some cases, however, the surfaces become ulcerated and the tubercular tumor undergoes further disintegration, and

finally becomes merged in the ulcerative process which attacks the deeper tissues.

As the septum has already been attacked by the morbid process, the loss of tissue now becomes evident by the destruction of the tuberculous granulation; usually, the greater portion of the cartilage is found to have been destroyed, while the bony septum always escapes. Although now the most conspicuous symptom of the clinical picture is the perforation of the septum, the granulations and nodules found at the edge of the perforation furnish a valuable diagnostic sign to distinguish it from perforating ulcer of the septum, in which the edges are smooth and sharply defined. This form of destruction of the septum is not followed by any alterations in the external nose.

The subjective symptoms, which consist in obstructed nasal respiration, are at first insignificant, but increase with the growth of the tumor. Their appearance is occasionally preceded by epistaxis. The formation of crusts is no part of the clinical picture as long as the integrity of the epithelium is preserved, but it appears as soon as ulceration has begun.

It is convenient to mention the so-called scrofulous alterations in the nose in connection with the tuberculomata, which Koschier, from their histologic structure, describes as tuberculoscrofulous lymphomata. Scrofula, as a separate process, has ceased to enjoy the recognition it formerly had, and is now generally regarded as a manifestation of tuberculosis peculiar to the childish organism. In addition to constitutional phenomena, it finds expression in chronic eczema, with infiltration of the skin at the anterior nares and the upper lip, where it produces the characteristic thickening of the scrofulous habit. The disease strongly resembles chronic dermatitis, for it is localized almost exclusively in those regions of the skin which are covered with epidermis, and it is at least doubtful whether we are justified in distinguishing the eczema of "true scrofula" from the form which often occurs in children as the result of nasal obstruction and consequent chronic rhinitis. Hence, scrofulous eczema does not extend beyond the plica vestibuli, which forms the boundary between epidermis and mucous membrane. Unless we can demonstrate the tubercular process in such infiltrations and erosions on the nose and upper lip, we can not consider scrofulous eczema as

a form of tuberculosis, and as this proof is lacking, and scrofulous eczema fails to show any peculiar characteristic, we can only designate it as a form of chronic eczema peculiar to the scrofulous habit.

On the other hand, those cases in which the tuberculo-scrofulous tumor shows a distinct tubercular structure are to be regarded as genuine manifestations of tuberculosis, in no way connected with scrofula ; such cases frequently go on to granulation and ulceration, with occasional destruction of the septum and of the inferior turbinated bone. Although in practice the conception of scrofula as a distinct morbid process may be expedient, it can not be denied that the term is often used to cover many processes in the childish organism for which as yet no satisfactory explanation has been found, and it is consequently advisable to restrict its application as much as possible.

The second or ulcerated form of nasal tuberculosis presents the characteristics of ordinary tuberculosis affecting mucous membranes. Infiltrations going on to degeneration, with the formation of ulcers with infiltrated edges and covered with granulations (Schech [1] stands alone in describing them as poor in granulation tissue), form the anatomic basis and lead to a more or less extensive destruction of the nasal mucous membrane, which can be demonstrated clinically. The ulcers vary in depth, and may spread to the bones and cartilages, where they lead to necrosis and deformities in the bony and cartilaginous framework.

In this form of the disease the principal symptoms are at first epistaxis, the formation of crusts of dried secretions, and the discharge of mucopus, so that it was formerly described as a tubercular ozena.

When the bone is involved, there might be some difficulty in distinguishing the condition from syphilitic disease, were it not for the fact that in every case of advanced nasal tuberculosis undoubted signs of tuberculosis are found in the lungs, for it appears from the observations published thus far that this form of tuberculosis always occurs secondary to extensive tubercular disease of the lungs.

Finally, there is a *third form of nasal tuberculosis, beginning in the bone or cartilage*, which Koschier [2] described on the strength of a single observation, although it is in accord

[1] "Krankh. der Mundhöhle," etc., Fifth Edit., p. 317.
[2] "Wien. klin. Wochen.," 1896.

with earlier descriptions by v. Volkmann, who observed this form of the disease quite frequently. Nevertheless, I am inclined to consider it much less frequent than the other two, especially the tumor-like variety, which I have often observed myself, while I have yet to see my first example of the former variety. I shall therefore quote the description given by Koschier, in which three factors are emphasized as characteristic of this form of the disease. These are, in the first place, alterations in the form of the external nose, which, as has been said, do not occur in the other forms; the wide distribution of the disease, which does not confine itself to one side of the septum, or even the entire septum, but takes in almost the entire skeleton of the nose; and, finally, the comparatively early appearance of large, deep ulcers in the mucous membrane, together with extensive necrosis, and the separation of necrotic portions of the cartilage and bone. These are the diagnostic points which serve to distinguish it from the variety of nasal tuberculosis which originates in the bony and cartilaginous portions.

Tubercular disease of the *pharynx* is infrequent. Tuberculomata on the posterior surface of the uvula (Avellis [1]) and on the roof of the pharynx (Koschier [2]) must be regarded as extremely rare. Mouret [3] described a unique case of tubercular granulations about the size of a bean appearing on the palatal tonsil of a patient twenty years of age, suffering from pulmonary and laryngeal disease.

The ulcerated form, first described by Isambert,[4] occurs more frequently. The anemic mucous membrane is the seat of closely aggregated grayish nodules about the size of a split pea, which later coalesce and break down. The ulcers, which have been minutely described and designated as "lenticular" by B. Fränkel,[5] are characterized by a tendency to grow toward the periphery rather than to invade the deeper tissues. The edges of the ulcer are slightly infiltrated and are irregular in outline, while the floor is covered with minute granulations and a dirty yellow secretion. In accordance with the superficial seat of the ulcers there is no diffuse infiltration of the mucous membrane,

[1] "Deutsche med. Wochen.," 1891, Nos. 32 and 33.
[2] *Loc. cit.*
[3] "Rev. hebd. de lar.," 1896, No. 54.
[4] "Ann. des mal. de l'oreille," I, 1875, p. 77, and II, p. 162.
[5] "Berlin. klin. Wochen.," 1876, No. 46.

such as is seen in the larynx. These ulcers are found chiefly on the soft palate and on the uvula; sometimes on the anterior and posterior arch of the palate and on the lateral pharyngeal wall; and in rare cases on the posterior pharyngeal wall and in the postnasal space. Although the ulcers are superficial, they nevertheless produce extensive destruction in the soft palate, but they have never been known to attack the bone. The subjective symptoms consist in dysphagia, and often in violent pain radiating toward the ears. In some cases partial cicatrization is said to occur. Kraus [1] reports having seen adhesions of the soft palate; but these accidents are rare, for there is very little tendency to spontaneous cure. Hence the prognosis in this form of pharyngeal tuberculosis, characterized by the presence of miliary nodules with a tendency to degenerate, is very unfavorable. The great majority of cases, as pointed out by Isambert, occur in the last stages of pulmonary phthisis, and this fact is of value in the diagnosis, which occasionally presents difficulties to the novice, who might be in danger of mistaking the tubercular for syphilitic ulcers.

Another manifestation of tuberculosis in the pharynx is seen in the cold abscesses which sometimes occur in the posterior pharyngeal wall, and are due to carious disease of the vertebral column. They give rise to a fluctuating tumor about the size of a hen's egg, usually on one side of the posterior pharyngeal wall, at a level varying with the particular vertebra affected. The patient complains of a sensation as of a foreign body in the throat when he swallows, and the voice has the well-known palatal quality. The presence of these symptoms of primary vertebral disease differentiates the diagnosis from tumors or other varieties of abscesses.

In the *larynx* we distinguish three forms of tuberculosis: One characterized by infiltration followed by degeneration; superficial ulcers (arrosion ulcers); and, finally, the tumor-like variety—tuberculoma. The most frequent form of the disease is the first-mentioned, and it presents such typical phenomena that the diagnosis can, as a rule, be easily made from the characteristic infiltration and ulceration. As the infiltration is due to the formation of tubercles in the mucous membrane, and has its seat in the submucosa,

[1] "Nothnagel's Handbuch," XVI, 1. Th., 1. Abth., p. 276.

the clinical picture varies with the anatomic relations of the mucous membrane in the various portions of the larynx, the degree of swelling depending on the thickness of the submucous tissue ; and according as the mucous membrane is or is not in close relation with the other structures in the larynx, especially the cartilage, there will be a greater or less tendency for the infiltration and ulceration to spread to these deeper parts. On the plica vocalis, where there is no submucous tissue, the stage of infiltration is less conspicuous than it is in the interarytenoid space, where the looser structure of the submucous tissue presents a favorable medium for the development of infiltration and secondary edema. Where, as on the epiglottis and the arytenoid cartilage, the mucous membrane is closely adherent to the cartilage, or in the vocal process, where it is intimately joined to the elastic fibers in the cartilage, the infiltration is very likely to extend to the perichondrium and to set up a perichondritis followed by necrosis of the cartilage ; while, on the other hand, if the disease is situated on the ventricular bands or the aryepiglottic folds, there is less danger of its spreading to the adjoining cartilages.

The most frequent seat of infiltration is the mucous membrane in the interarytenoid space,—*i. e.*, the interior surface of the posterior laryngeal wall,—so much so that its appearance in this situation is almost pathognomonic. In the early stages of the disease there is in this region a slight swelling, which becomes prominent when the mucous membrane is stretched, as in deep respiration. The swelling is not uniformly distributed over the posterior wall, but forms a slight prominence, either in the middle or to either side of the median line. It may be distinctly isolated, like a tumor, as Türck [1] described it, while the covering of mucous membrane remains intact. At first there may be some difficulty in differentiating these tuberculous infiltrations from chronic laryngitis, in which the parts are also swollen, especially when the entire upper respiratory tract shares in the descending catarrh ; the catarrhal swelling is, however, diffuse, being due to uniform thickening of the mucous membrane. The latter arches forward toward the interior of the larynx in the respiratory position, but in the median position becomes puckered into folds. The color

[1] "Atlas," I, XVII, vol. II.

of this catarrhal infiltration is characteristic, being a bluish-gray or whitish-gray, in consequence of the catarrhal thickening of the epithelial layers.

In the tubercular variety as the disease progresses the infiltration increases in size and its surface becomes nodular. At this time functional disturbances begin to appear. The accurate apposition of the arytenoid cartilages, on which normal function depends, becomes mechanically impossible on account of the tumor-like infiltration, and more or less pronounced hoarseness develops. The laryngoscopic image simulates the appearance of a paresis, as the posterior portions of the vocal cords fail to approximate on account of the swelling.

The epithelium itself now begins to undergo alteration. As the tubercular infiltrate approaches the surface the upper layers of the epithelium become necrotic and assume a grayish-white discoloration, the surface finally undergoes more extensive alteration, and we have the formation of ulcers and granulations.

The tubercular ulcers are characterized by elevated, infiltrated margins, which in the laryngoscopic picture largely obscure the floor of the ulcer owing to the foreshortening of all plane surfaces in the reflected image, so that the true condition is sometimes difficult to recognize. The second characteristic of tubercular ulcers is a tendency to the formation of granulations in the floor of the ulcer, and as it is difficult in ordinary laryngoscopy to see all of the posterior laryngeal wall, it is often impossible to determine whether there are deep ulcers or granulating surfaces hidden behind the infiltrations; it is, however, of little practical significance, as the granulations themselves rapidly undergo decomposition, and there is throughout the disease a continual alternation between granulation and ulcerative disintegration. Thus the surface presents an irregular appearance, ulcerating areas alternating with papillary masses of granulations, and, when seen in profile from above, suggesting the picture of a chain of mountains with narrow valleys running between them. It is well to bear in mind that the disease is usually more extensive on the posterior wall than appears in the laryngoscopic image. Whether the ulcers and granulations extend from the interarytenoid space down below the vocal cords, or occupy only the upper segment of the posterior wall, the laryngoscopic image will be

the same, as the elevated infiltrated margins of the ulcers completely hide the deeper portions. In such cases it is often possible to obtain an approximately correct image of the surface by employing Killian's method of examining the posterior wall, which consists in having the patient bend his head well forward while the operator sits on a low stool, or even kneels down in front of him. Even better than this is Kirchstein's method, which permits the observer to obtain a most satisfactory view of the posterior wall.

From the interarytenoid mucous membrane the morbid process extends to the posterior extremities of the vocal cords, which are eventually destroyed. Sometimes large flat ulcers extend from the posterior wall to the vocal cords, and if the granulations do not happen to be very abundant, these may easily be overlooked.[1] On the other hand, it must not be forgotten that the arytenoid cartilage is occasionally visible through the pallid mucous membrane above the vocal processes, and might in that case be mistaken for an ulcer. The epiglottis and the aryepiglottic folds are favorite seats for the tubercular process, and suffer the same destruction that we have described in the case of the posterior wall. The course of the disease can readily be traced on the epiglottis. The infiltration is the first change to appear, and lends a cushion-like shape to the epiglottis, which covers the greater part of the interior of the larynx. Later, ulceration begins accompanied by the appearance of granulations and grayish tubercles the size of a split pea in the neighborhood of the ulcer. If the aryepiglottic folds are involved there is usually marked swelling; the lateral wall of the larynx is attacked, and after the breaking-down of the infiltrated area this may lead to the formation of deep ulcers. The infiltrated ventricular bands become so swollen that they completely hide the vocal cords; occasionally ulcers and granulations are seen in the ventricle of the larynx, the former breaking directly through the ventricular bands into the interior, the latter projecting from the entrance like papillomatous tumors. Eventually, the tissues in all these regions of the upper portion of the larynx suffer more or less destruction, as the ulcers show little tendency to heal spontaneously by cicatrization, and the infiltration constantly tends to spread.

[1] See illustrations in Schnitzler's "Atlas," Plate IX, Nos. 1 and 2.

In the vocal cords the tubercular changes in the early stages are less pronounced, the catarrhal disease being more conspicuous than the infiltrations. The vocal cord is red and swollen, and assumes what is usually described as a cylindrical form. But even in these early stages the distribution of the disease, which often does not include the entire vocal cord or is confined to one-half of the larynx, points to tuberculosis rather than to catarrh, where the changes are usually symmetrical. In rare cases a series of tubercular nodules resembling a string of pearls is observed on the free border of the vocal cord. In most cases, however, the inflammation in the vocal cords is followed by destruction of the epithelium, and the formation of ulcers covered with a yellowish exudate. It is worthy of remark, as pointed out by M. Schmidt, that when the vocal cord is covered by a diffuse superficial ulceration, the yellowish exudate occasionally makes it appear almost normal. No matter how small or superficial an ulcer may appear in a tubercular patient, it should be regarded as tuberculous, as there can be no question of its being a catarrhal ulcer.

The tissue destruction that takes place in the subsequent course of the disease first attacks the free border of the vocal cord, and later extends over larger areas. It is frequently accompanied by active granulation, forming large masses resembling a cock's comb on the vocal cords, and in some cases leading to stenosis of the glottis. Sometimes the swollen and infiltrated vocal cord presents a furrow running parallel with and underneath the free border, converting the structure into two separate folds, one above the other. A picture of this kind is seen when ulcers appear on the lower surface of the vocal cord, or when there is a series of ulcers, above described as resembling a string of pearls.

The different forms of tuberculosis described thus far may vary in their extent and in the order of their appearance, and give rise to a great variety of clinical pictures. When, however, the infiltration extends to the cartilaginous frame of the larynx, the appearance changes, infiltration and ulceration of the perichondrium being followed by necrosis of the cartilage. The epiglottis and the arytenoid cartilages with their vocal processes, being nearest to the favorite seat of the disease, are most frequently attacked ;

more rarely, perichondritis extends to the crico-arytenoid articulation, and from there to the arytenoid and cricoid cartilages, the thyroid cartilage being very rarely involved.

Whenever the epiglottis shows signs of edema, perichondritis should be suspected. The peculiar structure and porosity of the epiglottis, which permit the glands and blood-vessels to pass through the cartilage from the laryngeal to the oral surface, allow the infiltration to spread in all directions, so that we do not get necrosis of the cartilage, but rather a complete liquefaction. This progresses *pari passu* with the infiltration of the mucous membrane, and may end in complete destruction of the epiglottis. In addition to the edema which characterizes the disease in the epiglottis, there is the symptom of pain, usually described as radiating toward the ears. Sometimes the dysphagia becomes so great that the taking of food gives rise to excruciating pain.

The infiltration, as has been said, is prone to spread from the posterior wall to the posterior portions of the vocal cords, where the vocal processes present a favorite seat for the disease. In this situation redness and swelling first appear, sometimes without involving the ligamentous portion of the vocal cord, so that the inexperienced observer is led to suspect pachydermia. Soon, however, deeper ulcers appear in these regions, and microscopic examination shows that there is a disintegration of the reticular portion of the arytenoid cartilage. Later, the process spreads to the perichondrium of the hyaline portion of the cartilage, and thus secondary perichondritis is followed by necrosis of the cartilage and the separation of sequestra.

Perichondritis of the arytenoid cartilage produces a characteristic swelling and edema in the aryepiglottic fold, and motion is impeded solely by the mechanical pressure of the swelling. Before long, however, the disease spreads to the capsule of the crico-arytenoid articulation, and, after destroying the joint, attacks the cricoid cartilage. This results in interference with the movement of the arytenoid cartilages, which finds expression in an apparent paresis of the vocal cords. Although it has been said that an edematous swelling over the affected portion of the cartilage is an important diagnostic point, it may be well, in order to avoid a misunderstanding, to point out that it has diagnostic value only when it is preceded by the break-

ing down of infiltrations in the areas mentioned, so that if a larynx is seen to be affected in this way at the first examination, there is always a possibility that one has to deal with a simple tubercular infiltration of the mucous membrane. Deep ulcers in the aryepiglottic folds are very often surrounded by edematous areas; perichondritis of the cricoid and thyroid cartilages is rare and presents no typical appearances. The diagnosis of the tubercular nature of the disease is based on the appearances in the other portions of the larynx. There are rare cases in which there is a so-called external perichondritis, the morbid process appearing on the external surface of the cartilage, principally on the lateral plates of the thyroid.

Lastly, we may mention three symptoms which are occasionally described as characteristic of tubercular laryngeal disease: anemia of the laryngeal mucous membrane, catarrhal laryngitis, and paresis of the vocal cords. *Anemia of the mucous membranes* is an expression of the general phthisical habit, and can not be regarded as a symptom of beginning laryngeal tuberculosis.

The question whether or not there exists a tubercular catarrh of the larynx is difficult to decide, and there are experienced laryngologists who believe it to be possible; but in those cases where the laryngoscope shows an uncomplicated image of catarrhal laryngitis it is more scientific to speak of chronic catarrh of the larynx associated with tuberculosis of the lungs than to speak of tubercular catarrh, since the latter term is hardly justified by the clinical and anatomic appearances. Paresis of the vocal cords is a symptom that frequently occurs in the beginning of tuberculosis and occasionally forms the prelude to tubercular disease; sometimes it appears only periodically after excessive use of the voice. E. Fränkel[1] found that it was due to atrophy of the muscular fibers, but the question whether tubercular changes occur in the muscle so early in the disease, or whether we have to deal with simple fatigue of the muscle due to anemia, such as occurs in all grave organic anemias, can not be determined at present.

There is another variety of tubercular ulcers differing from those following infiltration, which we shall describe as *arrosion ulcers*, due to local tubercular infection by the

[1] "Virch. Arch.," LXXI, p. 261.

sputa. They are distinguished by their superficial character and their tendency to spread over the surface of the membrane. Their favorite seat is the epiglottis, especially its free border; after that, the surface of the larynx, the aryepiglottic folds, and the lateral wall of the larynx. They begin as small ulcers the size of a split pea, with a moderately injected base, and finally become covered with necrotic epithelium, which separates and exposes a shallow depression. The ulcers run together and tend to spread toward the periphery, so that eventually large areas of the mucous membrane become involved. They occur principally in the later stages of pulmonary tuberculosis, and are found usually combined with other tubercular appearances in the larynx.

A rarer form of tuberculosis is found in the tuberculomata, which appear as circumscribed tumors. We learn from an exhaustive analysis of the cases by Avellis [1] that they grow most frequently "in the ventricle of the larynx under the angle of the glottis and on the posterior wall; more rarely on the ventricular bands; and least frequently on the vocal cords." Panzer [2] reports three cases of tubercular polyps on the vocal cords from Chiari's polyclinic. These tumors frequently do not differ from ordinary fibromata of the larynx, and, as a rule, show no tendency to ulceration. In some cases they must be regarded as a primary localization of the tubercular process, as no signs are found in the lungs or other organs of the body; in such cases their true nature can be determined only by histologic examination, for they are absolutely without any clinical characteristics. The prognosis is good if the tumors are removed; M. Schmidt [3] remarks that he often observed removal of the tumors to be followed by permanent cure or by a long period of health, until a new ulcer or a hemorrhage of the lungs supervened and confirmed the microscopic diagnosis.

TUBERCULOSIS OF THE EAR.

While the manifestations of tuberculosis in the larynx, though varying in their external appearance, are funda-

[1] "Deutsche med. Wochen.," 1891, Nos. 32 and 33.
[2] "Wein. med. Wochen.," 1895, Nos. 3–5.
[3] "Die Krankh. der ob. Luftwege," 2d edit., p. 362.

mentally the same, this is not the case with the organ of hearing. In the larynx the diagnosis is readily made, even in advanced stages of the disease, by the presence of infiltration, ulceration, and granulation ; but in tuberculosis of the ear the clinical picture varies greatly, and there is no characteristic course. This may be partly explained by the fact that the aural disease at first presents no more alarming symptoms than difficult hearing and discharges from the ear, and does not come under observation until quite late, when the process is so far advanced that it can not be distinguished from a simple chronic otitis media. Hence it is that the most prominent features of the picture are destruction of the tympanic membrane, suppuration and abscess formation in the mucous membrane of the middle ear, and carious destruction of large portions of the temporal bone, which separate as sequestra, while the granulations, which are so characteristic of the tubercular process, are comparatively insignificant.

It is almost superfluous to say that nowadays we base a description of tuberculosis of the organ of hearing exclusively on the demonstration of tubercle bacilli or on the histologic appearance characteristic of tuberculosis. We merely mention the fact because even in recent times such authors as Bezold and Hegetschweiler depend on the macroscopic appearance of the clinical picture and neglect bacteriologic examinations. That Bezold and some other authors differ from most of the authorities in regard to the diagnostic significance of tubercle bacilli in suppurative aural disease is due to the fact that there is a want of agreement in the literature as to the presence of tubercle bacilli. Among forty cases of otorrhea in tuberculous subjects, Nathan found tubercle bacilli in only twelve instances, while Lucae was unable to find them once among seventeen patients whom he had inoculated with tuberculin. On the other hand, I have rarely failed to find the bacilli, although I have examined a large number of cases.

But this failure to demonstrate the bacillus in every instance is explained, as already pointed out by Gottstein, by the fact that the pus is derived from the tubercular carious foci in the middle ear, which, it is well known, often fail to show the presence of bacilli. When we consider that, as Krause [1] has shown, the finding of bacilli in tubercular

[1] "Tuberculose der Knochen und Gelenke," Leipzig, 1891, p. 7.

bone disease is rare, and when we consider also that we often fail to find bacilli in undoubted cases of pulmonary tuberculosis, it can not surely be denied that the finding of bacilli should be a deciding proof of the existence of the disease. It is possible, by using proper methods of staining, to avoid the errors which are sometimes occasioned by the smegma bacillus. The latter is often found in old, purulent foci. Brieger supposes that the tubercle bacilli found by Bezold in cholesteatomata were really smegma bacilli, and I have myself found them in the pus derived from a sarcoma of the ear; they were easily decolorized with alcohol or dilute hydrochloric acid.

Tuberculosis of the ear may occur in any stage of the pulmonary disease, but it presents itself most characteristically in the later stages. It may be unilateral or bilateral, although some authors maintain that the left ear is more often affected than the right. A universal characteristic of tuberculosis of the ear is the absence of pain, which often leads the patient to neglect the disease as unimportant, so that the earlier stages do not come under observation.

It would appear that tuberculosis in the ear is usually secondary. The few cases so far reported as primary are open to criticism, and for the present we have no proof of primary tubercular osteomyelitis of the mastoid process.

It is difficult, if not impossible, from the clinical point of view, to decide whether one has to deal with primary tuberculosis of the bone, with secondary involvement of the tympanic cavity, or with the opposite condition; accordingly, we find that opinions are divided on the subject (Küster and Schwartze). But it would seem plausible to assume that we have to deal with primary tuberculosis of the bones of the ear in those cases in which there is a diffuse tubercular bone disease with fistula formation in scrofulous children.

Chronic tuberculosis, which is the most frequent form, is probably due to infection by way of the lymphatic channels. Barnick [1] supposes hematogenous infection in chronic tuberculosis of the middle ear to be quite frequent, especially in scrofulous children, "in whom, after rupture of a cheesy focus containing a few bacilli, the infection carriers are transmitted by the blood."

[1] "Arch. f. Ohr.," vol. XL.

Next in order of importance as a channel of infection we have the Eustachian tube. As the mucous membrane of the tube shares in the general atrophy characteristic of the phthisical habit, the lumen is usually dilated, and readily permits the entrance of sputum from the postnasal space. If there is ulceration in the nasopharynx, the conditions are, of course, even more favorable for infection. This mode of infection is perfectly possible, since the bacilli are capable of penetrating between the epithelial cells of the tympanic mucous membrane, even when the external layer of the epithelium is intact. It is further supported by the fact that tuberculosis most frequently begins in the middle ear. On the other hand, it would appear, from E. Fränkel's[1] observations, that the danger of infection from the postnasal space is not very great, for among fifty autopsies of tubercular patients, he found ten cases of tubercular disease in the postnasal space, without implication of the ear.

We have no means of judging whether it is possible for tubercular disease of the middle ear to be produced by direct immigration of the tubercle bacillus through a tubercular infiltration in the tympanic membrane, as there are no facts to support such a supposition.

On the tympanic membrane tuberculosis attacks both the epidermis and the mucous membrane.

The former variety is rare, and lacks histologic demonstration ; it includes only those cases in which there was undoubted tubercular disease in the external layers of the tympanic membrane without involvement of the middle ear. There is so little material that it is impossible to describe any distinct form for the tuberculosis ; the descriptions by Stacke and Preysing (from Körner's clinic) differ widely, so that one is forced to assume two distinct types, a miliary, nodular form and one which appears as a granulation tumor. In Stacke's case [2] the tympanic membrane is described as presenting a bulging of its posterior half, and a yellowish discoloration, as though there were an exudate behind it. The surface was covered with split-pea-sized yellowish nodules, with small vessels radiating from their centers. The tympanic cavity contained no exudate. The redness of the tympanic membrane gradually increased,

[1] "Zeitschr. f. Ohr.," x. [2] "Arch. f. Ohr.," vol. xx, p. 270.

and a yellowish ulcer formed in the inferior posterior segment and led to a gradually increasing perforation. The examination failed to show the usual tubercular appearances.

Preysing,[1] on the other hand, described a case of multiple tubercular tumors on the skull, in which the tympanic membrane was perforated in its inferior anterior segment and presented a flesh-colored, uniform mass, which eventually proved to be tubercular granulation tissue. The author got the impression "that the tympanic membranes became infiltrated and broke up into tumor-like masses, but the external layer of the epidermis was always found to be intact." The typical form of the disease in the tympanic membrane is that which begins in the mucosa and ends in the destruction of the membrane.

According to Habermann's histologic investigations, the disease begins with the formation of tubercular nodules appearing on the tympanic membrane as small, yellowish elevations, which rapidly break down and lead to liquefaction necrosis of the entire membrane, although some cases are found in which the mucosa alone is involved, while in a few others the substantia propria also shares in the process. As the nodules break down, the integrity of the membrane is destroyed, so that we often see several distinct perforations at the same time, before the coalescence of the ulcers has resulted in the entire destruction of the membrane. Hence, the statement of various authors that tubercular perforations preferably affect the inferior posterior segment of the tympanic membrane can not be accepted.

A significant diagnostic point is the painless course of the disease; while other inflammatory processes in the tympanic membrane are always associated with great pain, the symptom is almost always absent in tuberculosis in spite of the wide-spread inflammation and tissue destruction.

The nodule formation followed by disintegration is also characteristic of the disease in the other portions of the middle ear, but it is evident from the paucity of the reported cases that it is rarely possible to demonstrate it clinically. Usually, tuberculous disease of the middle ear presents itself in the guise of chronic otitis media. There is an

[1] "Zeitschr. f. Ohr.," XXXII, p. 369.

abundant discharge of a seropurulent secretion, which may be more or less offensive. As the result of the breaking-down of the caseous nodules, the mucous membrane presents an ulcerated appearance. Granulations and polypi are not present, as a rule.

As we have said in connection with the perforation of the tympanic membrane, the course of the disease is usually painless, but, on the other hand, it is characterized by rapid impairment of the hearing. This is due to the extensive infiltration, which spares neither the walls of the middle ear nor the immediate adjacent parts. One of the chief characteristics of the disease is the rapid development of caries, which soon destroys the ossicles and bony walls of the middle ear. Later, it involves the labyrinth and the mastoid process and leads to extensive tissue destruction, followed by grave functional disturbances. Eventually, the walls of the carotid artery and jugular vein may be eroded and fatal hemorrhage result, or the facial nerve is destroyed and paralysis ensues, or there may be total deafness as the result of the destruction of the labyrinth. There is a constant danger of the process spreading to the interior of the skull, and thus producing fatal results. Either the carious bones undergo cheesy degeneration and are cast off as sequestra, or they merely show a gray or black discoloration. Marked granulation is usually absent. The entire bone crumbles, and it is often possible to remove large sequestra with the forceps. The operator is often surprised to see how deep the destruction has gone, especially in children, often without any external signs; not rarely it is possible to remove the entire bony wall as far as the middle or posterior fossa, so that a large portion of the interior of the skull is laid bare. Necrotic portions of bone from the labyrinth or the posterior wall of the auditory meatus are occasionally discharged through the ear. In neglected cases we often find behind the ear a bone fistula that continues to discharge for many years.

LUPUS.

Lupus of the mucous membranes of the upper air-passages may be primary or secondary to lupus of the external skin. The primary form appears most frequently in the nose, although in recent years cases of primary disease

in the pillars of the fauces, base of the tongue, and larynx have also been reported. But the secondary form is far more frequent; it coexists with lupus of the external skin, being communicated to the interior of the nose, the upper lip, or the external lip, especially the alæ; or, from the skin surrounding the mouth, to the mucous membrane of the cheeks, the palate, the pharynx, and the larynx.

The chief characteristic of the disease is its painless course, which explains why the primary lupus eruptions on the mucous membranes usually escape observation, and the patient does not present himself for treatment until he is made aware of his malady by functional disturbances or by beginning deformity. This is abundantly proved by the systematic rhinoscopic and laryngoscopic examination of all cases of lupus of the external skin, for, according to Chiari and Riehl's [1] statistics, the larynx was involved in 6 out of 68 cases of lupus of the skin—*i. e.*, in 8.8% ; while in former years, when an examination was made only when demanded by the subjective symptoms of the patient, only 6 out of 725 cases, or 0.8%, were found to present this complication.

Lupus of the mucous membrane presents the characteristic reddish-brown nodules, as large as the head of a pin, slightly excoriated or covered with silvery epithelial scales which run together and form extensive, slightly elevated infiltrations, or even grow into larger masses of a distinct papillomatous appearance. As in the external skin, the lesions show a marked tendency to break down, and the resulting scars lend to the diseased areas their well-known appearance. "Occasionally, the disintegration of contiguous infiltrated areas leads to the production of deep ulcers, which, owing to successive granulations, fail to heal, and are followed by the formation of irregular, glandular masses of hypertrophic and disintegrated granulations, traversed by bands of cicatricial tissue" (Chiari and Riehl). In spite of the active ulceration there is little tendency on the part of the ulcers to attack the deeper structures, and the bony skeleton of the nose and the cartilaginous structure of the larynx, excepting the epiglottis, are not as a rule involved; on the other hand, the destruction of the cartilaginous septum is a frequent, not to say regular, phenom-

[1] "Vierteljahrsschr. f. Dermat. u. Syph.," 1882.

enon. The scars show a tendency to contract, and this produces stenosis at certain points on the entrance to the nose, on the isthmus of the fauces, on the entrance of the larynx, and on the vocal cords.

As regards lupus of the nose, it is found most frequently in the vestibule, and spreads from there to the septum, to the floor and lateral walls, and to the turbinated bodies. The external nose appears swollen at the tip and about the alæ, and shows deformities corresponding to the cutaneous destruction, for the cartilaginous and fibrous portion of the septum may be destroyed without the exterior of the nose being attacked by the disease. The nose becomes swollen and drops forward, the tip coming nearer the upper lip. The most striking deformity is seen in the septum; at first, while any tissue remains of the dividing wall, it appears on inspection to surround a huge perforation; but later, when this slender remnant of tissue disappears and the nose loses its support, it is converted into a mere pendulous mass; finally the ulceration attacks the remaining tissues of the nose, or the formation of cicatrices leads to further distortions and deformities.

The cartilaginous septum occasionally presents a form of lupus described by the French as "lupus pseudo-polypeux."[1] It often appears isolated, without any coexistent lesion in the external skin, but without a reservation we can not accept this as a special form of the disease, as there appears to be good reason to include it among the tuberculomata.

Lupus of the mucous membrane preferably attacks the uvula, the pillars of the fauces, the posterior and lateral pharyngeal walls, and especially the base of the tongue, whence the disease may spread to the epiglottis and to the larynx. In fact, the epiglottis is the point of election, and, according to Chiari and Riehl, escaped in only 3 out of 38 cases. Next in order of frequency follow the epiglottic folds, and last the vocal cords. It has been stated that the ulcers show no tendency to involve the cartilage (Kaposi,[2] however, as against this generally accepted view, mentions a case of complicated laryngeal perichondritis and chondritis), with the sole exception of the epiglottis,

[1] Comp. Simon in "Rev. de lar., d'ot.," etc., 1895, No. 17.
[2] "Lehrb.," 4th edit., p. 776.

the cartilaginous matrix of which falls an easy prey to the ulcerative process.

The papillomatous granulations or cicatricial adhesions occasionally lead to laryngeal stenosis which may demand tracheotomy. Lupus of the vocal cords produces disturbances in the voice. On the whole, however, the course of the disease, especially in the initial stages, presents no symptoms, and the patients are remarkably free from pain.

Lupus of the external ear differs in nowise from that of the external skin, and needs no special description.

2. LEPROSY.

Although leprosy is not endemic in our part of the world, an accurate knowledge of its nature is nevertheless necessary, as we not rarely meet with sporadic cases, especially in the large cities where there are many foreigners. We find it stated in the latest descriptions of the malady that the mucous membranes of the upper air-passages, as well as the external skin, are a favorite seat of the disease, so much so, in fact, that the peculiar raucous voice produced by laryngeal involvement has been regarded as absolutely typical of leprosy. Even at the present day there are so many more reports of leprosy of the larynx and pharynx than of nasal leprosy that after reading the usual text-books of the special literature one might get the impression that there is no typical clinical picture of nasal leprosy. The alterations that take place in the external tissues of the nose are well known, but the various descriptions of leprosy of the interior of the nose differ widely. It was customary to speak of epistaxis, "obstructive catarrh," perforation of the septum, and the development of ozena [1] as characteristic of the disease; and some authors describe a diffuse swelling of the entire mucous membrane, with the formation of nodes, which later became ulcerated on the surface. Until recent years opinions diverged in regard to whether the disease extended to the cartilaginous and bony skeleton. Virchow's [2] proposition

[1] Jeanselur et Laurens, Soc. méd. des Hôpit. u. Lepra-Confer., Berlin, 1897. Joseph, "Berlin. klin. Wochen.," 1896, No. 25. "Zwillinger und Läufer, "Wien. med. Wochen.," 1888, Nos. 26 and 27. "Journal of Laryng.," 1888, No. 1 (M. Mackenzie).

[2] Geschwülste II, p. 520.

was that "true perforation of the septum and depression of the bridge of the nose do not occur." This statement is not borne out by clinical experience. Although it is probably true that these destructions are not due directly to leprous disease of the cartilage and bones, yet necrosis, such as follows all ulcerative or inflammatory diseases of the nasal mucous membrane, undoubtedly does occur, as the mucosa plays a very important part in the nutrition of the skeleton of the nose, and even to some extent replaces the periosteum.

In recent years the study of the pathogenesis of leprosy has produced a number of new theories in which the nasal alterations play an important rôle. The question was discussed at some length in the Leprosy Convention held in Berlin in 1887, and since that time opinions in regard to the significance, frequency, and time of appearance of the nasal disease have undergone a marked change.

In the older literature we frequently see it stated that the nose becomes involved later than the larynx and pharynx, and it was believed that the nasal disease was secondary to the pharyngeal process, although this theory is in direct opposition with the assumption that leprosy is an infectious disease which does not spread by continuity, but by the extension through the lymph-channels of the leprous infiltrate to all parts of the skin and mucous membrane.

We now know that the nose is frequently attacked before the deeper portions of the air-passages, or may even be the only seat of the disease. Indeed, according to Glück's [1] statistics, the percentage of nasal leprosy is greater than that of leprosy of the larynx and pharynx. Again, opinions differ as to whether the process in the mucous membrane is secondary to the cutaneous eruption or is to be considered as going hand in hand with it, as it was believed that the mucous membrane was not involved in the leprous process until a later stage of the disease had been reached. At the present time, however, there is a general conviction that the process in the mucous membrane is a concomitant of the cutaneous disease, or even precedes it. After Glück had published his percentage of 89.19 in a series of 33 cases, Lima and de Mello found the frequency of early appearance of leprosy in the nose to be 95.83%, and

[1] Berl. Lepraconfer., 1897, 1, 1. Abth., pp. 19, 20.

thereby placed leprosy of the nose in its proper light. The question was finally solved when it was pointed out that in most cases the *first effects of the infection were to be found in the nose, and that the nasal secretions of lepers constituted the most important factor in the spread of the disease.*

Sticker[1] in a careful examination of 153 lepers "failed to find distinct anatomic changes in the nose in only 13 cases," and of these 13 there were 9 whose nasal secretions contained numerous bacilli of leprosy, thus affording another strong argument in favor of the view we have just stated, which is that of Sticker and of some others. We find a similar difference of opinion in regard to tuberculosis of the nasal mucous membrane, which has been found very much more frequently since its appearance was demonstrated clinically ; it now plays a considerable part in the pathology of the nose, and is universally regarded as the result of direct infection. In both cases the divergence is explained by our advance in the knowledge of nasal diseases, as, owing to the polymorphous nature of the interior of the nose, early alterations are not very characteristic, and are merely classed under the head of chronic catarrh. When the disease has progressed so far that the changes are clearly visible in the skeleton and outer covering of the nose, the diagnosis becomes extremely easy, but the analysis of the changes becomes more and more difficult as the disease progresses, and the clinical picture becomes more and more complicated.

As an example of the clinical picture seen in the early stages of leprosy I may mention a case which I had occasion to examine in the medical clinic of Professor Curschmann. It agrees so perfectly with the descriptions given by Glück, Zwillinger, and Läufer of the earliest appearances in nasal leprosy that I will give it in lieu of a general description :

A man, thirty-six years old, who had lived in Brazil since his sixth year, developed signs of anesthetic leprosy during the last three years, and during the last six months patches and diffuse infiltrations appeared on the skin, evidently the beginnings of a tubercular leprosy. The patient did not complain of subjective symptoms in the upper passages ; there was neither epistaxis nor nasal obstruc-

[1] Berl. Lepraconfer., 1897, I, 1. Abth., p. 99, and II, p. 55.

tion. There was a diffuse infiltration in the mucous membrane of the septum; the surface was smooth. As the result of the swelling in the middle of the right lateral wall of the nose there was a horizontal furrow, which at first sight looked like a deviation, while on the left side the hypertrophy was uniform. The mucous membrane over the turbinates was slightly hyperemic. The spongy tissue of the inferior turbinate was tense and swollen, returning to normal on the application of cocain, showing that these parts were not as yet involved in the leprous infiltration. The vibrissæ were preserved, the epithelium intact except in a small spot about the size of a split pea, situated in the previously mentioned furrow in the septum, at the boundary between the cartilaginous and bony portions. This area presented the appearance of a superficial ulcer, with smooth edges, not raised above the level of the surrounding mucous membrane. The secretion of the nose was mucopurulent and moderately abundant in both halves of the nose; the right side showed a greater tendency to the formation of crusts than the left, but the secretions did not possess any other characteristics of ozena. Thus, while the appearances of the interior of the nose were comparatively unimportant and not at all characteristic, the postrhinoscopic image presented very conspicuous signs of the disease. Here there was also a diffuse infiltration of the mucous membrane of the septum and of the upper margin of the choanæ, but, in addition, there were elevations about the size of a pea, with broad bases. These elevations were spotted, of a shiny yellowish color, corresponding to the whitish coloration of the nasal mucous membrane described by Lima and de Mello. The process did not extend beyond the posterior nares.

The leprous nature of these alterations was abundantly proved by the finding of innumerable leprosy bacilli, partly in clumps, partly in chains, or in the form of leprosy cells; and even in sections taken from the extirpated mucous membrane of the septum bacilli were found in large numbers.

Falling of the vibrissæ, which usually occurs late, was not observed in my case. There were no disturbances of sensibility in the nose. The sense of smell was somewhat impaired, although the patient himself did not observe it, and the examination with strongly odoriferous substances was difficult, as the man had lived in the primeval forests

since his sixth year, and was, therefore, unacquainted with the odors of any of them. In a series of 13 cases, Glück did not find much alteration in the sense of smell; the sensibility of the mucous membrane was reduced in 3 cases out of 6. Epistaxis is an almost constant early symptom of nasal leprosy, but in my case it had not occurred. As a rule, the mucous membranes are dry, and there is a tendency to crust formation, so that the picture of ozena is simulated. If the infiltration extends more deeply, and especially if it attacks the turbinated bones, obstruction of the nose and consequent interference with respiration result. The tendency of the nodes to break down is especially characteristic of the disease in the nasal mucous membrane, and in a short time ulcers develop in every part of the nasal cavity. As in the case of tuberculomata, septum perforations may be produced in the cartilage by the leprous infiltration. Lima and de Mello,[1] who give an excellent description of nasal leprosy, emphasize the predilection of the nodes and ulcers for the turbinated bodies, "which may be atrophied and porous, or partly or totally destroyed, so that scarcely a trace of them remains" (Glück). Defects have also been found in the other bones of the nasal skeleton, the vomer, the nasal bones, and the nasal spine; however, they are not to be regarded as due to leprosy, but rather as the expression of a disturbance in the nutrition of the mucous membrane leading to atrophy and caries of the bones.

In the pharynx leprosy affects principally the pillars of the fauces, the uvula, and to some extent the tonsils and the hard palate. Ulceration is said to be particularly apt to occur in the postnasal space. The fauces and the uvula become the seat of slightly elevated infiltrations, grayish-white or bluish in appearance, which undergo ulceration and cicatrization and lead to the formation of adhesions, particularly of the uvula. Perforations of the hard palate are mentioned by Zwillinger and Läufer, although Glück finds no confirmation of the statement in the literature or in his own cases. It is somewhat remarkable that Bergmann frequently found the posterior laryngeal wall intact when the other parts were affected with leprosy, and this is confirmed by Glück, although the latter adds that he

[1] "Monatsch. f. prakt. Dermat.," vol. VI, 1887, No. 13 and 14.

often found characteristic changes in this structure when the alterations in the mouth and nose were quite inconsiderable.

In the larynx the epiglottis is the commonest and earliest seat of leprosy. "It becomes uniformly hypertrophied and studded with nodules, and presents a characteristic form and position, being markedly displaced backward and more or less compressed from side to side" (Bergengrün).[1] "In severe grades of the disease the cartilage has a plump appearance; the lateral margins of the glottis are uniformly thickened where they merge into the aryepiglottic folds. In severer grades, where the uniform hypertrophy extends below the ventricular bands and involves the posterior laryngeal wall, the lumen becomes circular in outline; and, finally, in the severest grades the nodules on the thickened ventricular bands and the enormously hypertrophied posterior laryngeal wall reduce the lumen of the larynx to an orifice no larger than a lead-pencil, and completely obstruct the view of the deeper portions."[2] Both the ventricular bands and the vocal cords participate in the nodule formation and in the general hypertrophy, and even the subglottic mucous membrane is often markedly thickened. As the result of these changes, the voice becomes rough and hoarse; in the later stages laryngeal stenosis makes its appearance, and sometimes during the night produces attacks of suffocation, so that the leprous wards are constantly filled with the blowing, gurgling, and whistling noises of the occupants (Bergengrün). "The flat and comparatively extensive ulcers which form in the infiltrated and nodular portions of the larynx show a marked tendency to undergo cicatrization, and, as a result, not only is the mucous membrane sometimes shrunken and deformed, but even the cartilages may be reduced in size" (Glück). The cartilages of the larynx are never attacked alone, but the perichondrium almost regularly shows marked infiltration, and the bacilli may invade the cartilage from above (Neisser and Glück). Little is known as to the sensibility of the diseased mucous membrane in the larynx and pharynx. A few observers refer to the ease with which a laryngeal examination can be made, and attribute it to anesthesia of the parts.

[1] "Arch. f. Laryng.," vol. II.
[2] Schrötter, "Vorles. über Kehlkopf krankh.," 1892, p. 170.

In the external ear, characteristic changes, consisting in infiltration and nodule formation on the lobe, appear almost regularly in leprosy, but there is little mention in the literature of involvement of the organ of hearing. Extension of the leprous infiltration to the tubes may lead to tubular catarrh. Lima and de Mello [1] examined the ears of 48 lepers. They never found any alterations in the auditory meatus. In some cases the tympanic membrane was thickened, of a dull whitish color resembling "a fibrous plate," and "immovable during Valsalva's experiment." "There are adhesions in the wall of the tympanic cavity or between the ossicles." Other changes, consisting in anomalies of position and in convexity, are not leprous in character. In one case perforation of the tympanic membrane occurred; it was due to purulent otitis media.

3. MALLEUS HUMIDUS. GLANDERS.

Glanders is a disease of domestic animals, usually transmitted to man by the horse. The infection is carried by the malleus bacillus, which is contained in the secretion of the ulcers, especially in the nose, and gains entrance to the human organism through the skin or mucous membranes. The mucous membranes of the upper air-passages always share in the morbid process and present the characteristic miliary nodules, which later undergo ulceration. The breaking-down of neighboring nodules results in the formation of large ulcers, the floors of which are covered with a foul, sanguineous secretion. The ulcers show a marked tendency to invade deeper structures and occasion great tissue destruction.

Clinically we distinguish a chronic and an acute form. Chronic nasal glanders, according to Koranyi,[2] is ushered in by a feeling of fullness in the nose, a dry cold in the head, a feeling of heat in the throat, rough voice, cough, and, finally, by a mucous secretion, sparingly streaked with blood. Dry, blackish crusts are later expelled from the nose, and the mucous membrane underneath is seen to be swollen and ulcerated. Although the destructive process is slower than in the acute form, in the end the amount of

[1] "Mon. f. prakt. Derm.," 1887, p. 650.
[2] Nothnagel, vol. v. Part 5, p. 73.

tissue destroyed is quite as great. The acute form may occur either immediately after an infection or during the course of a chronic attack. It is accompanied by a cutaneous eruption, which spreads to the mucous membrane; often it reminds one of variola, appearing first in the form of red patches, which later are replaced by pustules (Koranyi). By extension of the ulcers which result from the breaking-down of the infiltrate large areas are destroyed; in the nose the septum becomes perforated, in the larynx the cartilaginous structure is destroyed. The voice is rough or hoarse as the result of erosion of the vocal cords; in the larynx the edema accompanying the ulceration sometimes leads to stenosis.

The disease attacks the nasal bones, and these, as well as the skin covering them, are destroyed. The accessory cavities of the nose are also involved. In Weichselbaum's [1] case masses of pus were found at the autopsy in the antrum of Highmore and in the frontal sinuses, and the mucous membrane was covered with numerous confluent yellow infiltrations. Occasionally, the disease attacks the cartilaginous orifices of the tubes. The prognosis of nasal glanders is fatal, both in the chronic and in the acute form, while in chronic glanders of the skin cicatrization of the ulcers and arrest of the malady have occasionally been observed.

4. FOOT-AND-MOUTH DISEASE. (THRUSH; STOMATITIS APHTHOSA EPIDEMICA.)

The mode of transmission of foot-and-mouth disease from animal to man has been extensively investigated in recent years. Although the disease almost exclusively concerns the digestive tract, if we disregard the constitutional phenomena to which it gives rise, it deserves to be mentioned in this connection, as it has also occasionally been observed on the mucous membrane of the nose, of the pharynx, and of the larynx. Koranyi [2] has given us a detailed description of the disease; Siegel, in various papers, has reported an epidemic and the bacteriologic examinations which it occasioned, and his results, while attacked

[1] "Wien. med. Wochen.," 1885, No. 22.
[2] Nothnagel's, "Spec. Path. u. Therap.," v, Part 5.

by some, are confirmed by Bussenius,[1] who agrees with Siegel in regarding a smooth, ovoid bacillus as the common cause of stomatitis in man and of foot-and-mouth disease in cattle. The typical lesions consist of blebs, which rapidly collapse and leave an ulcer, the floor of which is covered with a milky white exudate, while the edges are raised, dark red, and irregular in outline. The lesions are found on the tongue, gums, and palate, and occasionally also on the pharynx, and especially on the *free border of the epiglottis*. In the case reported by Bussenius large ulcers were found at the autopsy over the right arytenoid cartilage and on the epiglottis. According to Siegel,[2] the disease is often followed by "catarrh of the tubes," which "in children is usually purulent." Further details concerning this complication are wanting, but it is probably to be regarded as a suppuration from the middle ear.

5. ANTHRAX.

In the form of anthrax known as ragpickers' disease, or pulmonary anthrax, which is produced by the inhalation of dust containing the spores of anthrax, and which is usually ushered in by a chill, we find, according to Koranyi, who quotes H. Eppinger in support of his statement, "the nasal mucous membrane swollen, suffused with blood, and the seat of small carbuncle-like formations; the mucous membrane of the pharynx red and swollen, the tonsils covered with a diphtheroid membrane, the epiglottis red in color and hypertrophied."

6. ACTINOMYCOSIS.

Infection with actinomyces occurs usually in the mouth and oral pharynx by the wounding of the mucous membrane with spicules of grain, and is then followed by the well-known infection of the jaw and the submaxillary region. In rare cases actinomycosis extends to the larynx (Mündler[3] and Bérard[4]). There is in all cases a board-like infil-

[1] "Arch. f. Laryng.," VI, 1897. [2] "Arch. f. Larnyg.," III, p. 181.
[3] "Beitr. zur klin. Chir.," 1892.
[4] "Lyon méd.," 1895, April 21; see in "Semon's Centralb.," XII, p. 320.

tration of the outer tissues of the neck, which in Bérard's cases formed a rigid collar embracing the entire middle portion of the neck and rendering movement of the head impossible. The larynx, the pharynx, the thyroid gland, and the large vessels and nerves were found embedded in a mass of infiltrated tissue. Later, as a result of softening of the infiltration, pustules are formed, through which the characteristic yellow nodules are discharged.

In other cases the disease manifests itself in the formation of tumors on the thyroid cartilage and interior of the larynx; Störk [1] saw a tumor which involved the aryepiglottic fold, the arytenoid fold, and the epiglottic pharyngeal region of one-half of the larynx, and cites a similar case observed by Illich.

7. RABIES (LYSSA).

The clinical picture of rabies in man is characterized by symptoms of irritation in the sensory and motor nerves, their intensity depending on the course of the disease. The hyperesthesia of the olfactory nerve finds expression in hallucinations of smell, that of the trigeminus in attacks of sneezing, both of which phenomena are observed in the prodromal stage of the disease. The full development of the disease is characterized by respiratory cramp, erroneously designated spasm of the glottis, which involves all the muscles of respiration. It is not even definitely known whether a so-called spasm of the glottis—that is to say, a closure of the glottis—occurs; in Pitt's case,[2] where a laryngoscopic examination was made during the attack, the glottis was found to be gaping, as a result (according to Semon) of violent irritation of the respiratory center causing contraction of the crico-arytenoideus posticus, the respiratory muscle of the larynx, and thereby effecting abduction of the vocal cords. Löri also examined a patient during an inspiratory spasm, and was unable to demonstrate any participation of the laryngeal muscles.

The implication of the auditory sphere manifests itself in a hyperesthesia of the auditory nerve.

[1] Nothnagel's "Spec. Path. u. Therap.," vol. XIII, Th. II, Abth., I, 2d vol., p. 169.

[2] Compare "Semon's Centralb.," I, p. 251.

8. TRICHINOSIS.

Navratil [1] and Friedreich [2] each reported a case of laryngeal paralysis, the result of trichinosis. The left vocal cord was immovable midway between phonation and respiration, and there was, in addition, a paralysis of the constrictors of the pharynx.

[1] "Berlin. klin. Wochen.," 1876, p. 292.
[2] Quoted by Löri, "Die Veränderungen des Rachens," etc., p. 237.

VIII. DISEASES OF THE KIDNEY.

The complications which may appear in the course of nephritis in the upper air-passages consist in edema, hemorrhage, and general nutritive disturbance in the mucous membranes.

Edema occurs in the pharynx and larynx, especially in portions where the submucosa is well developed. Edematous swellings are, therefore, found chiefly in the uvula, the posterior faucial pillars, and on the lateral pharyngeal wall; in the larynx they are constantly found on the aryepiglottic folds, either on one or on both sides, and in their extension to other portions of the organ obey the general principles governing the spread of edema in the larynx.

According to Fauvel and Schrötter,[1] edema of the larynx is often the first sign of nephritis, and therefore enjoys a certain distinction from a diagnostic point of view. It must, however, be a very rare occurrence; at least, Morell Mackenzie[2] failed to find a single case, although he made a laryngoscopic examination of 200 nephritic patients with this end in view. Löri[3] reports two cases observed by himself. The edema is passive, being entirely due to venous stasis, and is occasionally observed on the posterior tracheal wall. Appearances simulating laryngeal stenosis are seen in uremic conditions, for uremic asthma may resemble bronchial asthma if expiration is prolonged, or may simulate laryngeal stenosis if inspiration is prolonged (E. Wagner[4]). But the fact that it always appears periodically in individuals with normal respiration establishes the differential diagnosis. I once had occasion to make a laryngoscopic examination of this kind two days before the outbreak of a fatal uremia; in spite of the negative appearance of the laryngeal image, the apparent laryngeal stenosis had led the attending physician to perform tracheotomy.

[1] "Vorlesungen," p. 92, 1st edit.
[2] "Lehrb. übers. von Semon," I, p. 374.
[3] *Loc. cit.*, p. 80.
[4] Ziemssen's "Handbuch," IX, 3d edit., p. 70.

It may be mentioned that uremic *aphasia* [1] has been observed in combination with unilateral palsies, due solely to serous infiltration of the brain-substance.

A more familiar and more frequent occurrence than edema is *hemorrhage*, due partly to the increase in blood pressure and partly to the changes in the blood-vessels which are so frequent in chronic interstitial nephritis. In addition to the epistaxis, which is often severe, there may be lesser hemorrhages in the pharynx and larynx; they occur a short time before the appearance of the uremia. As in all diseases of the circulatory apparatus, we find nutritive disturbances in the mucous membrane, manifesting themselves as atrophic catarrh; and if at the same time there are similar hemorrhages, there usually results the form of nephritis and laryngitis which is sometimes described as the chronic hemorrhagic variety. In the case of uremia which I have just mentioned I found a remarkable appearance of the mucous membrane; there was a marked dryness throughout the upper air-passages, although there could not be said to be any atrophic alterations in the nose or in any other part. The nose, as well as the larynx, was covered with minute dark-colored coagula, the remains of hemorrhages in the nose and throat. These coagula were so completely dried out that they were expelled in the form of dust with the respiratory air-current, which, on account of the dyspnea, was very violent. Türck [2] describes a case of sudden hoarseness and pain in the larynx, in which the organ was much inflamed; at the autopsy the mucous membrane of the interior of the larynx was found to be red in color and covered here and there with patches of delicate croupous membrane. Löri [3] also observed a case, which he described as "diphtheritic," in the course of a chronic *parenchymatous nephritis:* "The lesions consisted in grayish-white crusts the size of a pea, embedded in the mucous membrane of the tonsils and of the left arytenoid cartilage, and in a similar more extensive alteration on the upper surface of the epiglottis, which disappeared in a few days." This clinical picture hardly justifies the diagnosis of "diphtheritis."

To Dieulafoye and his followers we owe a detailed description of nephritic aural diseases, a few cases of which are also

[1] Senator, Nothnagel's "Handbuch Nierenkrankh.," p. 69; and Jaeckel, Berliner Dissert., 1884.

[2] "Klinik," pp. 177 and 178, Case 20.

[3] *Loc. cit.*, p. 82.

found in the older literature. Morf[1] has contributed a comprehensive treatise, in which, in addition to three of his own cases, he discusses twenty-two others collected from the literature. He admits, however, that his explanation of the clinical appearances and the nature of the disease is somewhat faulty, as he classes into one group nephritic disturbances due to pathologic processes in the ear, demonstrable microscopically or by functional examination, and into another group cases in which it was not possible to account for the functional disturbances by any pathologic changes in the tissues.

It must be admitted that aural disturbances are rare in nephritis. The statistics reported are small as regards the number of cases, and not very reliable on account of the limited amount of material on which they are based. It can not be determined whether any one form of nephritis possesses any special power of producing disease in the ear, but it would appear that chronic diffuse nephritis is more apt to do so than any other; in a few cases aural disease was observed in chronic nephritis after intermittent fever and in scarlatinal nephritis. Clinical and anatomic investigations have shown that aural disturbances may occur in the course of nephritis as the result of the general edema, and in uremia after hemorrhages. Tinnitus aurium and loss of hearing also occur as the result of secondary disease of the blood-vessels; and, lastly, it may be mentioned that a certain influence on the development and course of purulent otitis media has been ascribed to nephritis.[2]

Hemorrhages in the middle ear have been described by Schwartze,[3] Buck,[4] and Trautmann.[5] They manifest themselves either as suffusions in the middle ear or as hemorrhages in the mucous membrane; the latter are explained by Trautmann as the result of diapedesis. The diagnosis can be made by the bluish-red coloration of the tympanic membrane seen in the otoscopic image, while the subjective symptoms vary according as the hemorrhage was sudden or gradual, the tinnitus aurium being accordingly rapid or more gradual in its onset.

As examples of the other form of impaired hearing due

[1] "Zeitschr. f. Ohr.," xxx., H. 4.
[2] "Die Krankh. des Ohres," pp. 185–188.
[3] "Arch. f. Ohr.," IV, p. 12.
[4] "Arch. f. Ohr.," VII, p. 301.
[5] "Arch. f. Ohr.," XIV, pp. 91, 92.

to general edema we may mention one case of Rosenstein[1] and two cases of Morf,[2] in which, in the course of a chronic nephritis after intermittent fever (twice), and in acute nephritis (once), a gradually increasing loss of hearing was observed culminating in total deafness. Here the tuning-fork test for air- or bone-conduction was almost or quite negative (Morf). In both cases the hearing was occasionally completely restored during the course of the nephritis, and such temporary improvement was always accompanied by improvement in the general condition and subsidence of the edema. Rosenstein's case is the only one in which an autopsy was held; it was entirely negative. The fact that the loss of hearing progresses *pari passu* with the development of the edema, the periodic complete return of the power of hearing, and its final disappearance, in the case of acute nephritis without any local treatment of the ear, suggests the explanation advanced by Rosenstein for his own case: that we have to deal with edema of the roots and trunk of the auditory nerve, and that the variations in the power of hearing are directly dependent on the increase or decrease of the edematous infiltration.

A similar explanation applies to the disturbance observed in chronic uremia due to serous infiltration of the brain-substance. It is impossible to find any anatomic changes to account for the tinnitus and loss of hearing, nor can the exact location of the lesion be determined by means of the functional test.

Many cases of tinnitus aurium and defective hearing occurring in the course of nephritis are undoubtedly referable to secondary disease of the blood-vessels, and belong in the same category with the phenomena observed in arteriosclerosis and valvular lesions.

Finally, we have to consider suppurations in the middle ear, which are considered by some authors—among them Morf, Voss,[3] and Haug—as caused, or at least influenced, by nephritis.

There have been observed in the course of nephritis acute and chronic catarrhal, acute and chronic purulent, and hemorrhagic inflammations of the middle ear, but the nephritic character of the aural disease has not been defi-

[1] "Nierenkrankheiten," 4th edit., 1894, p. 260.
[2] "Zeitschr. f. Ohr.," pp. 324 and 328.
[3] "Arch. f. Ohr.," XXVI, p. 233.

nitely established. In proof of its dependence on nephritis certain postmortem appearances have been cited, consisting chiefly in hyperplasia of the submucous tissue in the middle ear, interpreted as an edema; but such a finding is not in the least remarkable in view of the long duration of the cases under discussion (Gurovitsch [1] and Moos [2]), and is often found quite independent of nephritis. Morf claims that purulent processes in the middle ear have a remarkable tendency to produce necrotic osteitis and a carious liquefaction of the bony walls of the air spaces in the temporal bone, but his claim finds little support in the literature, and the autopsy in one case of purulent otitis media after nephritis, which suggests the possibility of a casual relation between nephritis and otitis (I mean that of Gurovitsch), did not show any marked disease of the bones. Although the suppuration had existed for three months, there was no more than a seropurulent fluid in the mastoid cells. Thus it is seen that there is not sufficient proof to warrant the assumption of a special nephritic purulent otitis. It would appear, however, from certain reliable observations, that the course of chronic otitis media may be influenced by a coexistent nephritis, any exacerbation of the renal trouble being accompanied by increase in the purulent flow, and vice versa. This interdependence is clearly shown in Gurovitsch's case, and it also appears, from observations by Voss, that the dyscrasia which accompanies nephritis is capable of aggravating an existing aural trouble in diabetes. The value of Voss' observations is somewhat impaired by the fact that they refer to a case of scarlatinal nephritis, since the development of both diseases—the nephritis and the otitis—depends on an intoxication, and therefore a coincident increase in both sets of symptoms may be explained by an increase in the common virus. Haug [3] mentions a case of scarlatinal nephritis and otitis in which opening of the mastoid process was followed by improvement in the nephritic symptoms, while a subsequent exacerbation occurred in consequence of retention of pus due to granulations; this observation should at least incite us to more critical investigation of the literature in this respect.

[1] "Berlin. klin. Wochen," 1880, No. 42.
[2] "Schwartze's Handb.," 1, 538.
[3] *Loc. cit.*, p. 188.

IX. DISEASES OF THE SKIN AND OF THE SEXUAL ORGANS.

1. DISEASES OF THE SKIN.

The vestibule of the nose, as far as the plica vestibuli, is lined with epidermis, and is therefore attacked by the same diseases that affect the external integument. The most frequent disease affecting the vestibule, and with it the upper lips, is eczema, which leads to the same appearances in these regions as on the external skin. The nose may be either the primary or the secondary seat of eczema, for in scrofulous children, the subjects of chronic rhinitis, we frequently observe the development of eczema, which, as a result of the continual irritation of the nasal secretions, tends to spread more and more and to invade the face, while, conversely, general eczema of the external skin sometimes spreads to the vestibule of the nose.

We could mention a large number of skin diseases which extend into the vestibule of the nose; but as their diagnosis and treatment are the same here as on the external skin, with the exception of infectious processes such as lupus and syphilis, which are treated of elsewhere, they need not be discussed in detail in this place. There are certain diseases of the skin which in rare cases also lead to alterations in *the mucous membranes of the upper air-passages* analogous to the general process, although presenting certain differences in their appearance. Thus, there is a greater tendency to loss of epithelium and ulceration, due no doubt to the maceration which occurs in the mucous membrane as the result of the secretion, the moisture, and the warmth of the air-passages, so that, speaking generally, the mucous membrane shows defects and ulcerated surfaces, whereas the diseased epidermis of the skin remains as a protective covering in the form of scales and crusts.

For this reason a disease in the mucous membrane will in a few hours undergo certain peculiar changes and lose

the characteristic appearance of the same disease in the skin. The vesicle-formation of herpes and the formation of larger blebs characteristic of pemphigus are rarely seen, as the loosened epithelial cells are rapidly cast off, and thus the typical appearance of the disease is destroyed; in impetigo and erythema the superficial infiltration rapidly undergoes necrotic changes and is replaced by ulcers, so that all these diseases present the uniform picture of an exudative process followed by ulceration, and Seifert [1] and Schech [2] accordingly include them under one clinical picture, which, when it occurs in the larynx, is designated by the latter as exudative laryngitis.

Owing to the scarcity of complications in the mucous membranes of the upper air-passages, we are reduced to a few reports from other countries,[3] which would be even scantier were it not for the fact that affections of the oral cavity, particularly of the hard and soft palates, are often included among diseases of the pharynx. Among the skin diseases observed in the nose, the pharynx, and the larynx are herpes, urticaria, lichen, impetigo, and erythema. I decline to include miliaria (Löri [4]) and eczema of the throat (M. Schmidt [5]), because I do not regard either of these affections as anything more than an acute inflammation of the mucous membrane accompanied with unusual redness and swelling of the gland ducts, in no sense to be compared with eczema or miliaria of the external skin. In a general way, the seats of predilection of these diseases may be said to be the uvula, the posterior and lateral laryngeal walls, the base of the tongue, the epiglottis, and the upper margin of the larynx; it is quite possible that a predisposing factor for this particular localization is to be sought in the mechanical irritation to which these parts are particularly exposed during the ingestion of food. Herpetic eruptions, like those illustrated in Krieg's "Atlas," [6] occur in the pharynx and larynx, usually in combination with herpes labialis; occasionally, the eruption in these parts is

[1] "Heym. Handb. der Laryng. u. Rhin.," I, p. 448.

[2] "Münch. med. Wochen.," 1898, No. 26.

[3] I shall not quote the reported cases; they have been given by Schech, "Münch. med. Wochen.," 1898, No. 26; by Seifert, "Heym. Handb. der Laryng.," I, p. 448, and by Klemperer, "Heym. Handb.," I, p. 1286.

[4] "Die durch anderweitige Erkrankung bedingten Veränderungen," etc., p. 86.

[5] "Krankh. der ob. Luftwege," 2d edit., p. 531.

[6] "Atlas," Plate XXXV.

secondary to that on the lips. As the vesicles are deprived of their covering they frequently coalesce and lead to the ulceration of larger areas, as pointed out by Stepanow,[1] thus complicating the differential diagnosis from syphilis and diphtheria. Schrötter [2] says he has never seen herpetic vesicles converted into ulcers. According to him, the membrane which in a short time replaces the vesicle separates without leaving any alterations behind. The literature is particularly rich in cases of pemphigus in the upper air-passage, where pemphigus vulgaris, pemphigus foliaceus, and pemphigus vegetans have been observed. As an example of pemphigus vulgaris we may mention Schrötter's case,[2] in which there were periodic eruptions in the larynx, varying in extent and analogous to those on the rest of the body: "Thus the clear, transparent vesicle would appear on the epiglottis, and after a few hours the contents would become turbid, and finally yellow. The vesicle itself gradually contracted and collapsed more and more, and finally lay in folds on the surface, like a croupous membrane. The surrounding area was not specially inflamed." Thost [3] gives the following description of chronic pemphigus foliaceus with implication of the nasal, laryngeal, and pharyngeal mucous membrane. The diseased portions of the mucous membrane "became the seat of isolated whitish patches, irregular in shape, and varying in size from a split pea to a quarter of a dollar. These patches consisted of loosened epidermis, which appeared like a crumpled piece of paper or hung in shreds, and in the nose and postnasal space became dry and scaly, while the snow-white color changed to a dirty grayish-brown, or even reddish tint, if any blood was present. The surrounding portion of the mucous membrane showed marked reddening. The white epithelial covering was easily torn, and could be removed from the rete Malpighii without difficulty, exposing the red papillary layer, which bled when touched with a probe." According to Thost, the process heals without cicatrization in a short time, often within twenty-four hours, although Krieg, in his case of recurring pemphigus foliaceus, of which he gives several very good illustrations,[4] speaks of the cicatricial appearance of the palatal mucous membrane.

[1] "Mon. f. Ohr.," 1885, p. 237.
[2] "Vorlesungen über die Krankh. des Kehlkopfes," 1st edit., p. 62.
[3] "Mon. f. Ohr.," 1896, p. 165.
[4] "Atlas," Plate XXXVI.

Neumann,[1] in describing pemphigus vegetans, points out that in an analysis of 41 cases the primary seat of the disease was frequently found in the pharynx and larynx, and once in the nose.[2] In regard to diseases of the mucous membranes in erythema exudativum multiforme, we have cases reported by Lanz[3] and Schoetz.[4] Du Mesnil and Marx[5] give an example of impetigo herpetiformis (Uffinger[6]) and one of lichen ruber acuminatus, while lichen ruber planus is described by Marx.[7] Occasionally, the mucous membrane of the upper air-passages participates in urticarial eruptions, especially in the chronic relapsing form, where, as described by Cala,[8] the mucous membrane of the larynx may gradually become swollen and lead to asphyxia.

Diseases of the skin frequently spread from the external skin to the external ear and auditory meatus. Eczema is the most important, although herpes, impetigo, pityriasis, psoriasis, and pruritus also occur. It is, of course, of the greatest importance in the treatment to decide what form of otitis externa eczematosa is present, and whether there is suppuration of the middle ear with perforation of the membrane. As the diagnosis is often rendered difficult by the presence of a marked purulent secretion due to the eczema, and by the impossibility of inspecting the drum membrane on account of the great swelling of the walls of the meatus, it is well to bear in mind the symptoms which establish a diagnosis of purulent otitis media, without the macroscopic demonstration of perforation of the tympanic membrane. If the pus contains mucus, it is a sign that the middle ear is the source of the discharge; while pulsation of the pus in the external meatus and the presence of bubbles in the discharge are positive proofs of the existence of a perforation. Except in cases of moist eczema or of other processes associated with transudation, when the epidermic layer of the tympanic membrane becomes thickened and the membrane itself chronically inflamed, these skin diseases do not invade the ear-drum or the middle ear.

[1] "Wien. klin. Wochen.," 1898, No. 8.
[2] Riegel, "Wien. med. Wochen.," 1882, p. 274.
[3] "Berlin. klin. Wochen.," 1886, No. 41.
[4] *Ibid.*, 1889, No. 27.
[5] "Arch. f. Derm. u. Syph.," 1889, XXI.
[6] "Semon's Centralbl.," XI, p. 386. Lukasiewicz, "Arch. f. Derm. u. Syph.," 1896, vol. XXXIV.
[7] Würzburger Dissertation.
[8] "La Semaine Méd.," 1889, S. 346.

2. THE INFLUENCE OF NORMAL OR PATHOLOGICALLY ALTERED SEXUAL FUNCTIONS ON THE UPPER AIR-PASSAGES.

RELATION OF THE SEXUAL ORGANS TO THE UPPER AIR-PASSAGES.

Our knowledge of the relations existing between the male, and especially the female, sexual functions and the upper air-passages has recently been enriched by a number of important additions.

Even the earlier literature contains a few contributions on the subject of vicarious menstruation through the nose, swelling of the nose and coryza during the menses or during the sexual act, and epistaxis as a consequence of masturbation. It has, however, been reserved to the most recent times (Hack) to reduce these relations to a system,[1] although it seems to me that Fliess occasionally exaggerates, in his otherwise noteworthy and on the whole scientific work on the relations between the nose and the female sexual organs. Such relations exist even under physiologic conditions at the time of *puberty* and during *cohabitation*, and in the female during *menstruation*, the *menopause*, and *pregnancy*.

The most important pathologic condition is found in masturbation, although some influence is to be ascribed to gynecologic diseases, especially chronic endometritis and displacement of the uterus. In the last category of cases, however, it is difficult to tell to what extent the primary disease, or rather the hysteric condition of the patient which frequently accompanies it, can be held responsible for the sequels, which usually manifest themselves in the form of a nasal reflex neurosis. Examining the symptoms observed in the upper air-passages under the influence of sexual disease, we find that they consist in the main of phenomena referable to the vascular system, such as *hyperemia*, *swelling*, *exudations*, and *hemorrhages* in the mucous membrane.

The vasomotor system of the entire body is intimately connected with the sexual functions. Hence it is easy to understand that in any universal determination affecting the entire body the specialized vascular system of the nose

[1] John Mackenzie, "Johns Hopkins Hosp. Bull.," Baltimore, Jan., 1898.

should be more extensively implicated than other systems in the body. We must bear in mind the abundant blood supply of the nasal fossæ, and especially the erectile tissue embedded in the mucous membrane of certain portions of the turbinate bodies and of the septum. The presence of this tissue, which in its structure is analogous to the erectile tissue in the sexual organs, suggests the idea that it bears a certain relation to the sexual processes in the body, and it has been stated that the swelling in the nose is analogous to that which takes place in the clitoris and in the penis. There are, however, two considerations which tend to disprove the existence of such a relation: In the first place, the nasal phenomena must be regarded as exceptional; and, in the second place, while the anatomic structure of the erectile tissue in the nose in a general way resembles that found in the genitalia in the arrangement of smaller cavities on the surface and larger ones in the deeper tissues, it presents one important difference in the fact that the individual cavities possess muscular walls, which, under the influence of the sphenopalatine ganglion, may cause its contraction or dilatation. As we have previously explained, the position of the tissue in the nose shows that it is concerned solely with respiration; if it had any connection with the sexual function, it would be hard to understand why it is found in the respiratory portion of the nose and not in the olfactory.

We therefore consider the congestion which takes place in the nose as a mere local expression of a universal determination in a region particularly rich in blood-vessels. The mildest grades correspond to the hyperemia that accompanies any simple swelling and usually leads to periodic obstruction of one or both nares; the secretion of the swollen mucous membrane is increased, and in the end may even go on to hemorrhage. These hemorrhages appear to be usually diffuse, like carious hemorrhages in other parts of the body; they do not lead to tissue-destruction, and are usually derived from the turbinate bodies, although the septum may also be the source of a habitual epistaxis. In the sexual life of the woman such conditions, which, in consequence of the congestion, are generally associated with headache, are observed in the beginning, or even as prodromal symptoms, of menstruation; but they may also have a compensatory function when the menstruation is abnormal, and finally

may appear after the establishment of the menopause at the regular menstrual intervals.

We should call attention also to the observation of various authors that these phenomena may also appear in a diseased nasal mucous membrane, for Mackenzie states that in ozena the odor becomes more intense and the secretion more abundant during the menstrual period.

The so-called "erysipèle cataméniale" may also be included among the vasomotor reflex neuroses referable to the genital organs. It manifests itself in redness and swelling of the tissues about the external nose and of the organ itself.

There are certain reflex relations between definite regions of the nasal mucous membrane and the female genitalia. Although they may appear very obscure, and in Fliess' [1] description baffle comprehension, their existence, proved by a series of well-known facts, can not be disregarded. As long ago as 1884 we find in Kupper's [2] paper a warning against the use of the galvanocautery on the erectile tissue of the nose in pregnant women, on the ground that he twice saw it followed by abortion, and Schech goes so far as to say that pregnancy is an absolute contraindication to the use of the galvanocautery.[3] Fliess has shown by an extensive series of investigations in the gynecologic clinic of the University of Berlin that there are certain points on the anterior extremity of the middle and inferior turbinate bodies and on the *tubercle* of the septum—designated by him genital areas—through which some influence can be exercised on pathologic conditions in the female sexual apparatus. By cocainizing the "genital areas" the pains which accompany or follow the menstrual flow can be relieved and labor pains can be reduced to a minimum, while by cauterizing these areas permanent cure of dysmenorrhea may be achieved.

The pharynx, and especially the larynx, as well as the nose, may be the seat of congestions which can only be interpreted as derived from the genitalia. As during puberty, at the time of the so-called change of voice, the mucous membranes of the upper air-passages are subject to congestions, which are often the cause of the voice becoming easily tired, similar hyperemic conditions occur

[1] *Loc. cit.* [2] "Deutsche med. Wochen.," 1884, No. 51.
[3] Schech, 5th ed., p. 289.

during menstruation, during pregnancy, and in certain uterine affections, which from their effect on the singing voice are generally much better appreciated by singers than by physicians. Ruault [1] observed hemorrhages from the vocal cords accompanying the menstrual flow.

Sensory disturbances in the form of paresthesia and hyperesthesia, depending on sexual influences, have been described in all the mucous membranes of the upper air-passages; they manifest themselves in dryness of the throat, a feeling as of a foreign body, and desire to cough. They may be due partly to the hyperemia of these parts, but more particularly to the irritable condition which characterizes the entire nervous system at these periods. Irritation of the olfactory nerves, in the form of hyperosmia and parosmia, is sometimes observed. In speaking of asthmatic attacks as produced by disturbances in the genital region we approach perilously near the boundary-line between conditions due to sexual disturbances and coexistent hysteric phenomena, a boundary which is difficult to define in practice.

Finally, we must mention those phenomena which manifest themselves during the sexual development of the biody in functional disturbances of the voice. The most famliar of these is the change which occurs at puberty. It is a purely physiologic process, due to the increased development of the larynx, which occurs at this time and necessitates the adaptation of the muscles to the increased size of the organ. In most cases the change from the childish treble to the adult register takes place during the time of puberty without any marked disturbances, providing the voice, which at this time becomes easily hoarse and fatigued, is not unduly strained. Occasionally a slight hyperemia is observed in the vocal cords, but there is no abnormality in the movements of the larynx. The change of voice may be considered pathologic only when it lasts for some time and when the voice after puberty retains a childish or uncertain tone, without the character of a definite register. In the male this consists in a high, piping voice, which often changes suddenly to a deeper tone for a few words under the influence of emotion; or, if it changes to a higher register, gives out altogether; while in the

[1] See "Semon's Centralbl.," VI, p. 323.

young girl, as pointed out by Störck, it becomes abnormally deep and rough.

In the male this falsetto voice, which must be regarded as the effect of an abnormal prolongation of the voice-changing period, is designated as the eunuch's voice (Fournier); it may last for only a short time after puberty, or may, as shown by numerous cases, persist a greater length of time as a more or less ridiculous vocal anomaly. Although the condition causes the patient a good deal of annoyance, it, as a rule, readily yields to treatment.

There is no alteration of the laryngeal image, either in the form of redness or anomalies of motion of the vocal cords, notwithstanding Fournier's attempt to construct a series of clinical pictures. This is what we should expect if we remember that the eunuch's voice is the expression of a disturbance in the coördination of the laryngeal muscles, consisting in a failure of the mechanism to adapt itself to the dimensions of the fully developed larynx, and the persistence of a false register. By a judicious series of exercises, consisting mainly in training the voice to adhere to a lower key, a cure can usually be effected in a few sittings.

The vocal changes which accompany old age, and consist in roughness or shrillness, may be due to ossification of the laryngeal skeleton and to consequent changes in the vibrations.

RELATIONS BETWEEN THE SEXUAL ORGANS AND THE EARS.

Stepanow,[1] Eitelberg, and Gradenigo[2] have observed cases in which hemorrhages from the ears occurred either vicariously or coincidently with menstruation. In most of the cases the organ of hearing had been affected with chronic catarrh or chronic suppuration, and the power of hearing was more or less reduced during the intervals between the attacks. The hemorrhages from the ear usually occurred on the day before the appearance of menstruation, and, in the cases of menstrual anomalies, on the days on which the menses should have appeared. They were usually confined to one side, the same ear being affected in every attack. The amount of blood varied from two drops

[1] "Mon. f. Ohr.," 1885, No. 11.
[2] "Arch. f. Ohr.," vol. XXVIII, p. 82.

to quantities greater than that of a normal menstrual flow. The hemorrhage is usually heralded by a kind of aura, consisting in headache, slight vertigo, and tinnitus aurium. The region of the hemorrhage appears to be the tympanic membrane and the external meatus, especially the mouths of the cerumen glands on the posterior and upper walls. After the hemorrhage has subsided, nothing abnormal is usually found except a slight hyperemia of the gland ducts referred to, though in Eitelberg's case the tympanic membrane was the seat of petechiæ.

We may mention that certain observers have reported the occurrence of hemorrhages at the time of menstruation in cases of perforating chronic otitis media, associated with granulations. These cases are too obscure to be regarded as vicarious hemorrhages. The same statement applies to a few doubtful cases in which hemorrhage is said to have occurred in the labyrinth at the appearance of the menses. (Jacobson,[1] Koll[2].)

The changes in the auditory function during these vicarious hemorrhages from the ear are interesting. During the hemorrhage there is a uniform hyperesthesia of the auditory nerve for all registers, and a diminution in the electric reaction. The sensibility is reported in some cases as increased; in others, as abolished. At the time of menstruation tinnitus aurium is often observed; it is probably due to the hyperemia accompanying the flow.

Masturbation is said to aggravate an existing aural affection and to exaggerate a chronic catarrh or suppuration. It is sometimes given as the cause of subjective noises, which are probably an expression of abnormal irritability of the vasomotor centers.

The connection between *pregnancy* and the *puerperium* and chronic catarrh of the middle ear is so generally recognized among the laity that it is given as the cause of deafness in an abnormally large number of the cases, but the value of the patient's statement in this respect is much reduced when we find that in most cases it is possible to demonstrate objective alterations in the ear which can not possibly be referred to that physiologic condition of the female organism. If, as I believe we are justified in doing, we exclude all cases of obstinate catarrh of the middle ear,

[1] "Arch. f. Ohr.," XXI, p. 280. [2] "Arch. f. Ohr.," XXV, p. 88.

the cause of which can be demonstrated in diseases of the nose and pharynx, and cases of former purulent otitis media with remaining alterations in the middle ear and on the drum membrane, there remain only the forms of so-called chronic catarrhal otitis media without alteration of any kind in the tympanic membrane, and cases attended with tinnitus aurium. Bezold [1] has found that among 190 women suffering from this form of middle ear catarrh, 17.9% referred the beginning, or at least a subsequent aggravation of their deafness, to pregnancy or the puerperium. In some cases there was a successive deterioration in the auditory power at each pregnancy.

These auditory disturbances are no doubt closely related to the disturbances in the circulation to which the female organism is subject during the time of menstruation and pregnancy, and to the anemic conditions which follow the puerperal period. Thus we find that, analogous to the influence exerted by diseases of the circulatory system on the ear, anemia and hyperemia constitute important etiologic factors in the production of functional disturbances of the auditory organ. As the deafness and tinnitus aurium which occur during menstruation may be regarded as the result of the general determination, and as representing hyperemic conditions in the deeper portions of the organ of hearing, it is equally plausible that the chronic venous stasis and increased irritability of the entire nervous apparatus which characterize pregnancy should be capable of producing disturbances in the auditory function. Concerning emboli in the ear during the puerperium, and pyemic disease of the ear after puerperal fever, we have very few contributions.

3. GONORRHEA.

The occurrence of gonorrhea in the nose and in the pharyngeal cavity is now beyond dispute, and the many assertions made to the contrary in former times are wholly without foundation. These depended partly on theoretic speculation in regard to the mode of infection of mucous membrane covered with squamous and cylindric epithelium in gonorrhea. The rarity of nasal infection in comparison to the frequency of gonorrhea is to be

[1] "Arch. f. Ohr.," vol. XXV, p. 225.

attributed to the fact that the vestibule of the nose is lined with epidermis. Infection is usually due to uncleanliness in the use of handkerchiefs; the skin, however, opposes a natural barrier to the invasion of the virus. We find not only in the new-born, in connection with gonorrheal conjunctivitis, where infection takes place during birth, but also in adults, as the result of direct transmission to the nasal mucous membrane from other sources, a purulent rhinitis as the result of this mode of infection, the nature of which is proved by the bacteriologic demonstration of the gonococci (Miller [1]). I once had occasion to observe two cases of purulent rhinitis in the secretions of which typical gonococci were found within the pus-cells, occurring in two children of the same family, aged four and six respectively, who lived amid poverty-stricken and uncleanly surroundings and shared the bed of their gonorrheal mother.

In this connection it is interesting to note the possibility of gonorrhea being conveyed to the oral mucous membrane of infants (Rosinski [2]), where it manifests itself in the form of a whitish exudate; Cuttler [3] and Salzmann [4] each report a case of gonorrheal ulcerative stomatitis, the result of an infection contracted by coitus per os.

Occasionally arthritis may be localized in the articulations of the larynx and produce symptoms similar to those which occur in acute articular rheumatism. Liebermann [5] and Simpson [6] describe a disease of the crico-arytenoid articulation which appeared in connection with swellings in other joints after an acute gonorrhea. In one of these cases the left arytenoid cartilage was the seat of redness and swelling, most marked over the articulation, and this on sounding was found to be fluctuating. The left vocal cord failed in adduction. At the same time there were aphonia and violent pain in the region of the larynx, increased by pressure on the thyroid cartilage. Gradually the voice improved, and after six weeks the swelling disappeared, although the vocal cord continued slug-

[1] Störk, "Nothnagel's Handb.," XIII, 1st half, p. 86.
[2] "Zeitschr. f. Gynäk," 1891.
[3] See "Semon's Centralbl.," VI, p. 166.
[4] Kraus, "Nothnagel's Handb.," XVI, 1 Th., 1 Abth., p. 244.
[5] From "Soc. méd. des Hôpit.," 1873, p. 388, reprinted by Archambault, Thèse de Paris, 1886.
[6] From "Med. Rec.," July, 1889, reprinted by Lacoarret, "Rev. d. Laryng.," 1891, p. 398.

gish in its movements after the voice had almost regained its usual quality. In Simpson's case the swelling subsided more rapidly, but there also remained a sluggishness in the movements of the vocal cord on the affected side and in the region of the joint when the patient was discharged after eleven days. Lazarus[1] has described a new variety of gonorrheal disease of the larynx on the strength of a case of bilateral paralysis of the crico-arytenoidei postici in gonorrheal arthritis. As no alterations could be demonstrated with the laryngoscope in the arytenoid cartilages or in the mucous membrane of the interarytenoid space, and as there was neither tenderness nor pain in the cartilages of the larynx, the clinical picture of this form is clearly distinguished from that seen in the articular affections just described, and we must agree with Lazarus—although he does not dwell on these points in the differential diagnosis—in explaining his case as one of *gonorrheal neuritis*, the occurrence of which finds ample confirmation in the investigations carried out by Leyden.[2]

The localization of gonorrheal disease in the ear has never been reported; Flesch [3] believes that he once found gonococci in the pus derived from the middle ear of an infant. One of Fischel's [4] histories contains a note to the effect that, in a case of gonorrhea, tinnitus aurium was followed within twenty-four hours by complete bilateral deafness, but it is of little value.

4. SYPHILIS.

We are unable to devote to syphilis of the upper air-passages the space which its importance and frequency demand. The subject is fully discussed in all text-books on syphilis and in many special essays,[5] so that I shall refer only briefly to the most important points.

Primary sores are found in the nose in the region of the vestibule, which is accessible to infection by the finger.

[1] "Arch. f. Laryng.," v, p. 232. [2] "Zeitschr. f. klin. Med.," 1892.
[3] "Berlin. klin. Wochen.," 1892, No. 48.
[4] Fischel, "Prag. med. Wochen.," 1891, No. 11.
[5] Neumann, "Syphilis," Nothnagel's "Spec. Path. u. Therap.," 1897, vol. XXIII. Lang, "Vorlesungen," 2d ed., 1895. Gerber, "Syphilis der Nase u. des Halses," "Berlin, bei Karger," 1895. Seifert, "Deutsche med. Wochen.," 1893, 42, 44, 45.

They have been observed on the alæ and on the septum, and deserve mention because they may obscure a diagnosis in two different ways. When the sore is situated on the inner surface of the alæ and leads to marked swelling and redness of that region, there is at first, before the glands of the face and neck become enlarged and the induration surrounding the ulcer becomes apparent, a possibility of mistaking it for furuncle; and when the symptoms are fully developed, the lesion may be mistaken for a gumma. The secondary stage appears on the mucous membrane of the nose at the same time as on the external skin, but it occurs less frequently and presents fewer morphologic varieties. Erythema and papules are probably very rare on the mucous membrane, for opinions differ as to the possibility of their occurrence there; they are somewhat more frequent in the vestibule and, according to some, on the floor of the nose and septum. Lang [1] depicts a vegetating papule situated on the boundary between the epidermis and cartilaginous septum. One form of early syphilis in the nose is a peculiar catarrh, differing from acute catarrh by its insidious onset and by the character of the secretion, which is thick, though scanty. It may possibly be regarded as a specific erythema. The mild character of the symptoms and the fact that complete recovery takes place—for superficial ulcerations in the mucous membranes are very rare (Lang)—probably explain the scarcity of the reports about this form of catarrh.

The most important manifestations of syphilis in the nose belong to the tertiary stage. Both the lesions themselves and the defects and cicatricial contractions which result after they heal often require local treatment. The hereditary forms resemble the tertiary in their course.

It is well known that gummatous disease may appear under various forms and run a very different course in different cases. The circumscribed tumor-like variety is rare in the nose; when it does occur, it is most frequently localized on the epidermic and cartilaginous septum and on the alæ. According to Koon, Manasse,[2] and Kuttner,[3] one ought to distinguish as a special form syphilitic granulomata, which, however, can not be differentiated from tuberculomata either clinically or histologically, at least in those

[1] *Loc. cit.*, Fig. 56. [2] "Virch. Arch.," Bd. CXLVII, p. 32.
[3] "Arch. f. Lar. u. Rhin.," VII, 1898.

cases in which it is impossible to find either tubercle bacilli or cheesy detritus in the tubercle. They differ from the ordinary gummatous tumors by the presence of a pedicle or a broad base and by their greater vitality, as they show no tendency to central necrosis, and only a slight tendency to superficial ulceration. Manasse regards them as simple connective-tissue tumors, originating in the submucous connective tissue, and either pushing the epithelium before them or breaking through it.

Gummatous infiltration with chronic inflammation leading to hyperplasia of the mucous membrane is very common, and manifests itself in the form of a hypertrophic rhinitis—the coryza neonatorum of hereditary syphilis. Sooner or later the process goes on to tumor formation, but before that event occurs the disease may attack the perichondrium and periosteum of the cartilages and bones of the nasal skeleton, and lay the foundation for necrosis of the cartilage and sequestration of the bone. Referring to such cases, in which sequestra were found under the hypertrophic, intact mucous membrane, Sänger [1] and E. Fränkel [2] make the statement that the bony framework of the nose may become diseased independently of the mucous membrane. Sänger distinguishes three forms of bone disease, which he calls exfoliated necrosis following suppurating processes, rarefying luetic osteitis, or caries sicca, and rarefying and plastic osteitis. We also recognize a syphilitic chondritis in addition to perichondritis. As a result of all these processes we find the familiar defects in the soft parts and in the bones, producing the characteristic cicatricial contractions and distortions, and often leading to adhesions and stenosis.

While any part of the bony skeleton may be attacked by the disease, the median and lateral walls manifest a peculiar predisposition. In the septum the bony portion is chiefly involved, and the vomer, as well as the perpendicular plate of the ethmoid bone, may be more or less completely destroyed by the necrosis. The situation of these defects is of significance in the differential diagnosis from tuberculosis, which produces its ravages especially in the cartilaginous septum; although it not rarely happens that the syphilitic process involves the cartilaginous as well as the

[1] "Vierteljahrschr. f. Derm. u. Syph.," 1877, pp. 89 and 90.
[2] "Virch. Arch.," 75.

bony septum. We must take exception to the statement made by Schech[1] that syphilis preferably attacks the anterior cartilaginous portion of the septum. The detection of a perforation of the septum is sometimes difficult when the mucous membrane is still in the hypertrophic stage, or if the sequestrum, as often happens, separates from the posterior extremity of the vomer. Next to the septum the turbinate bodies are the commonest seats of the disease, and are sometimes partly or completely destroyed. The floor of the nose becomes involved, and large perforations, heralded by swelling of the floor, occur in the hard palate and sometimes afford a view into the nose from the mouth. Lang mentions extension of the disease to the lamina cribrosa, with following meningitis. Syphilitic caries of the ethmoid cells has been reported by Gerber, Lange, and Hellmann[2].

After a hypertrophic syphilitic rhinitis has passed through the stage of ulceration, the cicatricial contraction or the atrophy of the mucous membrane leads to a condition that is clinically known as atrophic rhinitis. Owing to its conversion into scar tissue, the loss of the ciliated columnar epithelium, the degeneration of the blood-vessels, and the disappearance of the glands and erectile tissue, the mucous membrane loses the power of performing its normal function; the dried secretions accumulate in the widened cavities of the atrophic organ, undergo decomposition, and lead to the formation of crusts. The offensive odor with which the condition is associated has given rise to the unscientific and misleading term ozena syphilitica. It appears from the investigations of Zukerkandl[3] that the mucous membrane of the accessory cavities may also participate both in the hypertrophy and in the subsequent cicatricial process.

While crust formation and fetor are rarely absent in old cases of nasal syphilis, they may also be found in the earlier stages of a variety of diseases. Crust formation is observed in any form of ulceration, while fetor always accompanies necrosis of the bone and is constantly present in atrophic rhinitis. The tissue destruction that takes place in the course of syphilis in the skin and in the cartilaginous and bony framework of the nose produces the

[1] "Die Krankh. der Mundhöhle," etc., p. 311.
[2] "Arch. f. Lar. u. Rhin.," III, p. 210. [3] "Anat. der Nase," vol. II.

most extensive alterations, which only are not visible with the rhinoscope, but also leave an indelible mark of the disease on the external appearance of the patient. The well-known syphilitic nose is the terror of patients, and, with the exception of lupus, there is no other disease capable of producing such frightful disfigurement. The commonest deformity consists in the so-called "*saddle-nose*," characterized by the sinking of the bridge of the nose and elevation of the tip, while the flattening that accompanies it appears to increase the transverse diameter. The nose as a whole is reduced in size, which is explained by Neumann as a molecular atrophy of the bone. Besides the absence of the nasal septum, this deformity may be produced by various causes. Defects in the cartilaginous portion of the septum do not alter the shape of the nose, even when the vomer is destroyed. When, however, the upper anterior portion of the perpendicular plate of the ethmoid bone, the posterior support of the two nasal bones, is destroyed, the sinking of these bones, which is further increased by the cicatricial contraction, produces a marked change in the shape of the nose. There are, however, cases of saddle-nose in which no such coarse destructions of tissue are found, and several theories have been offered to explain their formation. According to Moldenhauer,[1] it is produced by a cicatricial contraction of the connective tissue that unites the cartilaginous and bony portions of the external nose, while Neumann [2] mentions two novel physical factors, "the first of which consists in the loosening and partial destruction of the connections between the bony and cartilaginous structures, and the second in a difference of atmospheric pressure between the external air and that of the interior of the nose which accompanies every inspiration." "As long as the framework of the nose is intact and the musculature of the cartilaginous portion performs its functions, these two factors suffice to maintain the equilibrium during the decreased pressure which accompanies the inspiration. But as soon as these structures suffer a loss of integrity they are no longer capable of resisting the external pressure, and a sinking of the nose results in the direction of the increased external pressure."

[1] "Lehrb. der Nasenkrankh.," Leipzig, 1886.

[2] Nothnagel's "Spec. Path. u. Therap.," XXIII, p. 344.

The destruction of the bony, cartilaginous, and epidermic portions of the septum produces a characteristic deformity of the profile, consisting in a depression of the nose, which, deprived of its posterior support, becomes a mere mass of flesh overhanging the nasal cavity, and, in obedience to the laws of gravity, approaches the upper lip. Various other deformities may be seen as the result of destruction of other parts of the bony or cartilaginous framework of the external nose, among which we may mention one that is particularly common in the hereditary forms of syphilis, and in which, in addition to the saddle-shape, there is a complete flattening of the nose by destruction of the alæ and cartilages of the lateral walls, so that in profile the nose is not raised above the level of the face, and in the front view presents the appearance of two irregular, distorted openings, corresponding to the anterior nares, covered by a perforated plate of tissue.[1]

The pharynx is a favorite seat of syphilis in all its forms. The primary chancre is found on the palatal tonsils, which are much swollen, dark blue in color, and frequently the seat of superficial ulceration, while the submaxillary and submental glands are at the same time greatly enlarged. Infection may take place in a variety of ways—by direct inoculation during improper practices, by eating with infected forks or spoons, and sometimes even by surgical instruments. It is important to mention that a chancre on the tonsil may be mistaken for diphtheric tonsillitis, tonsillary abscess, carcinoma or sarcoma of the tonsils, or for a gumma. The erythematous and papular eruptions which occur on the faucial pillars, on the tonsils, and on the soft palate are so well known that their description may here be omitted.

They are never observed on the posterior laryngeal wall, but are occasionally seen in the postrhinoscopic image on the posterior surface of the uvula.

Condylomata resembling papillomata are sometimes seen on the hard and soft palate, on the pillars of the fauces, and on the tonsils, in the form of pale gray nodular excrescences.

The tertiary forms of acquired syphilis and *the various hereditary types* produce marked alterations in the pharynx.

[1] Some instructive illustrations are found in Rang, *loc. cit.*, Figs. 62–68.

To begin with the tonsillar space, we may mention the gumma infiltrations, tumors, and ulcerations that are usually found associated with diseases in the nose and in the oral pharynx. The diagnosis in such cases presents no difficulties. It is more difficult when the nasopharynx alone is diseased. The symptoms complained of by the patient are very vague: headache, depression, lassitude, loss of appetite, and occasionally earache—nothing that might point to an exact diagnosis; and the diseased focus may remain undiscovered until for some reason a postrhinoscopic examination is made. We then find ulcerations in the roof of the pharynx, in the neighborhood of the choanæ, and sometimes on the lateral pharyngeal wall, which are readily recognized as syphilitic ulcers by their irregular outline, sharp edges, and excavated floors covered with yellowish secretions. The disease may invade the periosteum and the bone, or there may be from the beginning a syphilitic osteitis, ending in necrosis and extensive destruction of the surrounding bony walls. If the disease is situated in the roof of the pharynx, part of the sphenoid bone, if on the posterior wall, parts of the cervical vertebræ, especially the atlas and axis, may give way and cause large openings into the vertebral canal, or ulceration and severe hemorrhage from the vertebral artery may occur.

A gumma on the posterior surface of the soft palate generally results in perforation of that structure, usually just below its attachment to the palatal bone, and leads to various deformities, according to the size of the perforation. If the tissue destruction is great, the soft palate is loosened from its attachment and drops down, so that if the perforation is situated in the middle line above the uvula, the latter may come in contact with the base of the tongue. In extensive ulcerations the entire uvula and large portions of the soft palate and faucial pillars may be destroyed; and as the disease is not limited to the soft parts, the palatal bone itself is often perforated, so that it is possible to obtain a view of the nose from below.

The syphilitic alterations in the posterior and lateral walls of the pharynx deserve special attention, as they may be mistaken for follicular catarrh or for a chronic hypertrophic catarrh of the plica salpingopharyngea (Neumann), if they appear in the nodular form or in the form of diffuse

infiltrations. The true nature of the disease is easily recognized by its tendency to cause rapid destruction of tissue.

Krecke[1] once saw two hard, spherical granulation tumors, the size of a pigeon's egg, on the posterior wall of the pharynx, which showed no tendency to break down, and disappeared on the administration of potassium iodid. They probably belonged to the same category as the granulation tumors described by Kuhn-Manasse.

As has been stated, the tissue destructions that occur in the course of tertiary or hereditary syphilis are of the greatest importance, and their practical significance is accentuated by the subsequent cicatricial contractions and adhesions, which may lead to marked functional disturbances. While, on the one hand, destruction of the hard and soft palates produces changes in the voice and difficulty in swallowing by making it impossible to effect a closure of the posterior nares, the cicatricial contractions, on the other hand, frequently lead to stenoses in the nasal pharynx, which embarrass nasal respiration, and rarely to a stenosis in the deeper portions of the pharynx, which interferes with the ingestion of food.

The scar that follows the healing of a specific ulcer on the mucous membrane has the same radiate appearance characteristically seen in the external skin after the healing of syphilitic lesions. Where there is a solid foundation, as on the posterior pharyngeal wall, the mucous membrane has a tense, glistening appearance, resembling tendon, while in the neighborhood of the isthmus the scars lead to distortions of the soft tissues. The symmetry of the posterior nares is destroyed, the uvula is drawn to one side or rolled on itself, and the palatal ridges are distorted almost beyond recognition.

Neighboring areas in the mucous membranes are frequently the seat of cicatricial adhesions, which are due to the tendency of the lesions to produce contact ulcers on opposed surfaces. Thus, we frequently see bands of adhesion uniting the posterior pharyngeal wall to the soft palate. The adhesion may be so extensive as to shut off the oral cavity completely from the postnasal space, or the adhesion may be only partial, leaving a chimney-like opening into the postnasal space, through which the secretions from the

[1] "Münch. med. Wochen.," 1894, No. 47.

nose trickle down into the pharynx, as there is usually a coexistent chronic fetid rhinitis. These adhesions may be visible at the first glance on ordinary inspection, but some of them are more obscure, and require a postrhinoscopic or laryngoscopic examination for their detection. Among these we include the adhesions which are seen when the soft palate is only partly destroyed, and which take the form of a horizontal diaphragm-like membrane between the posterior surface of the soft palate, near its attachment to the palatal bone, and the posterior pharyngeal wall, or those which lead to the formation of adhesive bands in the postnasal space between the roof of the pharynx and the swollen orifices of the Eustachian tubes, or between the latter and the margins of the choanæ or the posterior pharyngeal wall. Both these forms of postsyphilitic alterations occasion great discomfort, the destructive variety interfering with nasal respiration and lending a peculiar dead quality to the voice, while the cicatricial form, by involving the tubes, leads to certain disturbances in the hearing, to be discussed later. A rare form of adhesion is one which forms between the base of the tongue and the posterior pharyngeal wall.

Synechiæ between the soft palate and the posterior wall are of such frequent occurrence and give rise to such distressing symptoms that they often require operative treatment. In view of the tendency of the two divided portions of an adhesion to reunite, and thus oppose a serious obstacle to the success of the operation, it may be well to discuss briefly the conditions which explain not only the original formation of the synechia, but also its tendency to recurrence. When the soft palate performs its normal functions, and when, in obedience to the laws of gravity, it retains its perpendicular position and moves with every act of deglutition and phonation, there is small danger of the opposing surfaces becoming adherent, even when they are the seat of ulcers, as the constant movement of the soft palate would loosen any adhesive bands as fast as they formed ; but when, on the contrary, the soft palate, as the result of deformity or the distortion of syphilitic scars, is brought nearer the posterior wall of the pharynx and loses its normal mobility, the conditions for the formation of an adhesion are proportionately more favorable. Neumann [1]

[1] Nothnagel's "Spec. Path. u. Therap.," XXIII, p. 320.

has pointed out that "adhesions are especially liable to form when the faucial pillars are totally or partially destroyed, and when, owing to an antecedent syphilitic myositis, the palatoglossus, the palatopharyngeal, and the pterygo-, mylo-, glosso-, and buccolaryngeal muscles, as well as the middle constrictor of the pharynx, fail to act."

The hoarseness of syphilis, under the name of "*raucego syphilitica*," was formerly deemed of some importance by physicians, is still regarded among the laity as a frequent sign of an old infection. It is, therefore, surprising to learn from the statistics that syphilitic disease of the larynx is comparatively rare. Statistics based on dispensary work in diseases of the throat show a rather low percentage of laryngeal syphilis. According to Schrötter,[1] 8.7% among 35,826 patients; according to Rosenberg,[2] 3.6% (there were 58 cases of specific laryngeal disease among 16,000 patients in B. Fränkel's polyclinic); while other authors give somewhat larger percentages, based on shorter series of cases. It might be thought that this conspicuous infrequency of the disease is due to the notorious indifference of the patients, and to the fact that many physicians do not feel called upon to devote any special attention to it, as it disappears under general antisyphilitic treatment, were it not for the fact that the investigations by syphilographers, made with a view to determining the laryngeal complications, have yielded similar results. The most reliable analysis is that made by Lewin,[3] who, among 20,000 syphilitic subjects in his clinic, found 575 cases, or 2.9%, of laryngeal diseases, 13% of which were grave and 87% comparatively mild.

Secondary syphilis appears in the larynx in the form of erythematous and papular eruptions, going on to ulceration; while the tertiary stage, which often appears as early as one year after infection (Semond[4]), is represented by gummatous disease, which may manifest itself as a small nodular syphilid, as a diffuse infiltration, or as a circumscribed gumma. The symptom-complex of laryngeal syphilis further includes the ulcers due to the breaking-down of the gummatous tumors and to the perichondritis

[1] See Gerber's statistics, *loc. cit.*, p. 44.
[2] "Krankh. der Mundhöhle," etc., 1893; Karger, p. 306.
[3] "Charité Ann.," vol. VI, p. 538.
[4] "Centralbl. f. Laryng.," X, 203.

which follows as the result of extension to the cartilages. Finally, we may regard as sequels the scar formations and the chronic infiltrations and contractions which remain and lead to permanent functional disturbances in the voice or to marked stenosis.

The question whether we are justified in considering catarrhal disease of the laryngeal mucous membrane as an erythematous eruption is still undecided, in spite of Lewin's [1] paper advocating the recognition of such an erythema. For my part, I agree with that author, and believe that the condition usually designated as syphilitic catarrh is not a catarrh in the ordinary sense of the word and differs clinically from an ordinary catarrhal laryngitis.

It is characterized by a peculiar, dark, bluish-red or brownish-red (Lewin) discoloration, which makes it appear in the laryngeal image like a peculiar, one might almost say specific, hyperemia, especially as it lacks swelling and increased secretion, the ordinary symptoms of catarrh of the mucous membrane. Although it has been so described by certain French authors, the red discoloration is not such as to justify the designation of roseola or macular syphilid, being diffuse rather than circumscribed.

In some cases the erythema as we have just described it becomes covered with gray patches or rings elevated above the hyperemic mucous membrane (as illustrated by Schnitzler),[2] resembling the mucous patches of the soft palate. Although they are also observed on the epiglottis and on the aryepiglottic folds, their favorite seat is on the vocal cords. The superficial layers of the epithelium very soon separate and the patches are converted into superficial ulcers. The occurrence of flat and of acuminate condylomata on the laryngeal mucous membrane has been described. The former are due to hyperplasia of the papillæ, and appear as pale gray prominences with broad bases, slightly elevated above the mucous membrane, preferably situated on the free border of the vocal cords, on the epiglottis, and on the aryepiglottic folds. They rather resemble papules or opalescent patches, and the old name of condylomata would perhaps best be discarded, especially in diseases of the mucous membrane. This applies still more

[1] "Charité Ann.," vol. VI. [2] "Atlas," Pl. XII, 1.

to the so-called acuminate condylomata that have been described in the larynx, the existence of which, however, is denied by the majority of authors—Lewin, for instance, never saw a case of this kind. They can not be positively distinguished from the granulating edges of an ulcer, or even from connective-tissue tumors, such as fibromata, papillomata, and so on, as they do not yield to antisyphilitic treatment.

Among tertiary lesions, as has been stated, we distinguish the three forms of nodular syphilids, diffuse gummatous infiltration, and gummy tumor.

The first of these manifests itself in the form of small nodules, varying from the size of a pinhead to that of a split pea, closely aggregated or even confluent. Lewin remarks that their covering of mucous membrane, which is at first normal, gradually assumes a yellowish discoloration as the process passes into ulceration. This form, which is also found on the palate and in the pharynx, may be difficult to diagnose from lupus or tuberculosis in the absence of evident signs of syphilis in other parts of the body. It is true that the nodules show less tendency to the scar formation which in lupus appears coincident with the formation of ulcers, and the reaction in the surrounding areas is less marked, but these phenomena are all so variable that we are often driven to the test of antisyphilitic treatment. The subjective symptoms are of some diagnostic value, since syphilitic disease, as in the other mucous membranes of the upper air-passages, runs a painless course, while lupus, and especially tuberculosis, is associated with severe pain in the throat and with dysphagia.

The diffuse infiltrations and the gummy tumors represent different expressions of the same gummatous disease. The former are the more frequent; the latter, until recent times, were considered as very rare forms, although a hasty survey of the last volume of Semon's "Centralblatt für Laryngologie" reveals a goodly number of cases. While the diffuse infiltrations preferably affect the epiglottis and the aryepiglottic folds, where they lead to a diffuse swelling, covered with healthy, smooth mucous membrane, the gummy tumors may be found in any part of the larynx in the form of circumscribed spherical bulgings. They also occur on the ventricular bands and below the vocal cords, are frequently isolated, and may, as long as the mucous

membrane remains intact, be confounded with incipient malignant tumors, such as carcinoma or sarcoma.

With the exception of certain nodular varieties, which may perhaps be compared to the syphilitic granulation tumors (Kuhn-Manasse), these forms are rarely demonstrated by laryngoscopic examination, as they possess a marked tendency to undergo ulceration.

The ulcers vary in size and depth. Those which develop from infiltrations are wide-spread and flat, while those which follow gummata are deeper, and correspond in size with the gumma which they replace. Their boundaries are sharply defined, the edges are undermined, elevated above the surface, and thickened, while the surrounding area is the seat of a dusky red discoloration, more or less distributed over the entire larynx. "The floor of the ulcer is covered with a thin, grayish, creamy exudate, the removal of which reveals the whitish speckled appearance of the firm infiltration"—(Orth). The differential diagnosis from tubercular ulcers is based on the sharp edges, the speckled floor, the absence of nodules in the surrounding area, and the absence of any tendency to the formation of granulations, although there are cases in which the diagnosis can be decided only by a bacteriologic or a general examination. It must also be borne in mind that syphilis and tuberculosis are not rarely associated, as was pointed out in various papers by Schnitzler. Moreover, we learn from daily experience that even when the external appearance of the ulcer fails to afford any diagnostic points, the diagnosis may be inferred from its situation in the larynx. While tuberculous ulcers are preferably found on the posterior laryngeal wall, and on the posterior extremities of the vocal cords, syphilis affects chiefly the ligamentous portion of the vocal cords. While tuberculosis is frequently unilateral, especially when it appears in the vocal cords, the syphilitic ulcers are always bilateral, and very often distributed symmetrically on the free borders, being evidently produced by contact of opposed portions of the cords. Tubercular ulcers usually occupy the surface of the vocal cords, while syphilitic ulcers are situated on the free border, and give to it a dentated appearance. The ulcers eventually break down and lead to tissue destructions which differ greatly in extent. The appearance of the laryngeal image varies widely, as any one can convince himself by

glancing through Schnitzler's or Krieg's "Atlas." Edema is not characteristic of syphilis, as some authors maintain; it always depends on ulceration or on perichondritis. The latter may be primary or secondary, more frequently secondary, and develops in any case of deep ulceration of the cartilage; it is, of course, followed by necrosis and exfoliation of the diseased portions, and the resulting defects in the framework of the larynx may give rise to great deformities and malpositions. Syphilitic is much less frequent than tubercular disease of the cartilage.

As syphilis readily yields to specific remedies, unassisted by local treatment, it often leaves conspicuous alterations, due to cicatricial contraction or to connective-tissue neoplasms, while tuberculosis of the larynx, owing to its unfavorable prognosis, rarely comes under observation in the stage of regeneration. The scars, which vary in depth and size according to the ulcers that they replace, are stellate in form, and by their contractions often produce distortions in isolated portions of the larynx, so that not only the vocal cords and ventricular bands, but also the aryepiglottic folds and the epiglottis, may be so displaced by the contraction of the scar that the relations in the laryngeal image are much disturbed. Subjective symptoms are usually wanting. On the other hand, adhesions between neighboring parts are common, especially between the vocal cords, which are usually both ulcerated along their free borders, and therefore present a favorable seat for the formation of synechiæ. The cords in such cases are united by cicatricial membranes, which always begin at the anterior extremity and extend for a variable distance backward, interfering with the mobility and function of the cords, very frequently giving rise to severe dyspnea. The ulceration in the epiglottis is sometimes so great as to destroy one-half of the structure or one entire free margin, and if, as frequently happens, it is bent backward and an adhesion forms between it and the aryepiglottic fold, the lumen of the larynx becomes obstructed and serious embarrassment of respiration may result.

Hausemann[1] recently described a certain cicatricial lesion on the epiglottis that he often had occasion to observe at autopsy, having found it in 25 out of 42 cases

[1] "Berlin. klin. Wochen.," 1896, No. 11.

of syphilitic subjects. The epiglottis, from the frenulum to the upper border, was the seat of a process resembling the so-called smooth atrophy of the base of the tongue, first seen by Virchow and minutely described by Lewin.[1] We omitted this because it is of no value in clinical diagnosis. The effect of the lesion was to draw the cartilage forward so as to effect an anteflexion of the epiglottis.

Among permanent postsyphilitic alterations we must mention a diffuse hyperplasia of the mucous membrane, which may lead to extensive stenosis of the larynx and dyspnea, if it occurs below the vocal cords. We find it mentioned by Neumann[2]; and Eppinger[3] describes it as a fibroid degeneration accompanied by ulceration or cicatrization and producing a diffuse puckering of the mucous membrane, such as Türck described after his so-called "parenchymatous inflammation of the mucous membrane." Whether the hyperplastic condition of the squamous epithelium—which has been called, after Virchow, pachyderma laryngis—is due to syphilis is not definitely known, but it seems probable.

I once saw paralysis of the vocal cords (paralysis of the right posticus) in secondary syphilis, which yielded to antisyphilitic treatment. A few other cases are found in the literature. The most natural explanation for this occurrence is enlargement of the mediastinal or peritracheal lymph-glands exerting pressure on the nerves, as syphilitic neuritis of the nerve-trunk is unknown.

Syphilis of the ear is definitely known to occur only in those parts which can be directly inspected: that is, on the external ear, in the external auditory meatus, on the drumhead, in the region overlying the mastoid process, and, with the aid of posterior rhinoscopy, on the pharyngeal orifices of the tubes. The external ear presents all the alterations that are seen as the expression of secondary or tertiary syphilis on the external skin, and in the much-quoted case of Zucker[4] even a primary affection of the external ear was demonstrated. The manifestations on the skin of the external ear correspond in time of appearance and morphology with syphilis of the external skin. Thus, we find roseola, papules, and condylomata in the secondary nodular syphil-

[1] "Virch. Arch.," vol. CXXXVIII. [2] *Loc. cit.*, p. 40.
[3] "Handb. der pathol. Anatomie," Klebs, 7th ed., p. 123.
[4] "Zeitschr. f. Ohr.," IX.

ids, and gummata in the tertiary stage. The cases recorded in the literature are comparatively few, and confirm what we learn from statistical sources of the infrequency of these complications. The course of the papular form in the external meatus is remarkable; it was first carefully described by Stöhr[1]. The wall of the meatus at first shows a muddy, bluish-red discoloration; this is followed by swelling and diffuse redness embracing the tympanic membrane, in which Stöhr also observed similar muddy, bluish-red patches. A few authors (Kretschmann, Lang) observed papules on the tympanic membrane, described by Lang as pale, glistening patches, the size of a millet seed, over the processus brevis. In the auditory meatus the papules lead to excoriations; the walls become very much swollen, and there is a copious flow of bloody, purulent fluid. Later, these excoriated patches become the seat of excrescences which eventually lead to the formation of condylomata presenting themselves as villi or polypoid structures with small bases, either within the external meatus or protruding from the canal. According to Christinneck, there is a tendency to the formation of circular ulcers at the entrance of the external auditory meatus.

The diagnosis of these affections is based on the presence of constitutional syphilis, as they are very easily confounded with otitis externa eczematosa or with granulations due to some other cause.

Gummata have been described on the external ear (Hessler[2]), on the bony wall of the external auditory meatus (Brieger,[3] Habermann[4]); on the tympanic membrane (Baratoux[5]); in the mastoid process, both central (Schede,[6] Haug[7]) and in the periosteum (Pollak,[8] Brieger[3]); they present no special characteristics. These affections all yield to antisyphilitic treatment, but they leave scars which may produce marked stenosis of the external auditory meatus, or periosteal deposits and exostoses on the bony portions of the external meatus and on the mastoid process.

The pharyngeal orifices of the Eustachian tubes may

[1] "Arch. f. Ohr.," v, p. 130.
[2] "Arch. f. Ohr.," xx, p. 242.
[3] "Beitr. z. Ohrenheilk.," p 161.
[4] "Schwartze's Handb.," i, p. 277.
[5] "Rev. mens. de lar.," 1885, No. 7.
[6] Quoted from Kloos, "Schwartze's Handb.," i, p. 486, § 29, No. 14.
[7] "Arch. f. Ohr.," xxxvi, pp. 201, 202.
[8] See "Arch. f. Ohr.," xviii, p. 204.

share in the syphilitic process in a variety of ways; they may be the seat of primary syphilis in consequence of infection by a polluted catheter, or they may be attacked during the secondary and tertiary stages in connection with the postnasal space and become involved in the resulting cicatricial contractions and adhesions. The seat and the nature of the disease are easily demonstrated by a rhinoscopic examination after symptoms in the middle ear, retraction, opacities, difficult hearing, and tinnitus aurium have aroused the suspicion of tubular occlusion. Suppuration from the middle ear is common in syphilitic subjects. So far, our clinical and anatomic observations do not justify us in regarding it as a specific suppuration, since it has not been possible to demonstrate the occurrence of irritative syphilitic processes in the middle ear, although theoretically the existence of syphilitic disease of the middle ear seems plausible. "Authorities in the main agree that in acute and subacute simple, as well as in acute and chronic purulent, affections of the middle ear occurring in the course of syphilis, the nasal and pharyngeal disease plays an important rôle" (Bezold [1]). The same etiology may be assumed for suppuration from the middle ear in hereditary syphilis. Fournier,[2] it is true, says that these suppurations may constitute the primary manifestations of hereditary syphilis, and mentions the absence of pain as a characteristic symptom in such cases, but his observations are not satisfactory from an otologic standpoint, and do not carry much weight.

Exudative inflammation of the middle ear is mentioned by Schwartze [3]; and Kirchner [4] subsequently observed such a case, which was, however, complicated by the existence of ulcers in the nasopharynx. At the autopsy Kirchner found in the middle ear, besides a serosanguineous exudate, round-celled infiltrations, split-pea-shaped neoplasms in the bone, and a constriction of the blood-vessels, which he interpreted as a syphilitic endarteritis. Kirchner's case is, however, not very convincing, and it seems remarkable that in his microscopic investigations he did not take any account of the fact that the cadaver had

[1] "Arch. f. Ohr.," XXI, p. 260.

[2] Lectures on Late Hereditary Syphilis, translated by Kōrbl and Zeissel, 1894, p. 150.

[3] "Arch. f. Ohr," VI, 267. [4] "Arch. f. Ohr.," XXVIII, p. 172.

been in water several days, and that he found no post-mortem changes. Finally, a form of sclerotic middle-ear catarrh has been described as a consequence of syphilis. Gradinego [1] and Chambellan [2] assume a sclerosis of the middle ear, which the former explains as a parasyphilitic affection in hereditary lues.

There is a form of syphilis affecting the nervous apparatus of the organ of hearing the existence of which is based solely on clinical observation. During the tertiary, and even more frequently during the secondary, stage, a few weeks after the appearance of the skin eruption, the patient suddenly complains of severe headache and loss of hearing, which may go on to complete deafness within a few days; the condition is always accompanied by tinnitus aurium or other subjective noises or harmonic tones, sometimes with vertigo and vomiting, and Schwartze [3] adds to these symptoms a reeling gait in the dark. The disease is usually unilateral, occasionally bilateral. Otoscopic examination reveals no alterations, but the functional test shows that the lesion is in the nervous path: Rinne's test is positive, and when the tuning-fork is placed on the head the tone may suddenly change to the healthy side; frequently there is inability to hear high-pitched notes. Gradenigo [4] describes three different varieties, according to the course of the inflammation: a slowly progressing, a rapidly progressing, and one with apoplectiform onset. Fournier very correctly points out a similarity between the latter form and the loss of hearing in tabes, without, however, recognizing an etiologic connection for all cases.

In the hereditary form there is a disease of the inner ear analogous to that which occurs in tertiary syphilis. It occurs principally between the ages of ten and twenty (six to eighteen), and is frequently associated with interstitial keratitis and Hutchinson's teeth, although it is much rarer than the ocular disease; Fournier met with it in only 40 out of 212 cases. Gradenigo says that the power of hearing often varies from one day to the next, but except for this, and the fact that the disease is always painless and bilateral, it does not differ from the form seen in acquired

[1] "Arch. f. Ohr.," XXXVIII, p. 310.
[2] "Ann. des mal. de l'oreille," 1895, p. 267.
[3] "Chirurg. Erkrank. des Ohres," p. 376.
[4] "Schwartze's Handb.," II, p. 424.

syphilis. There is, however, a marked difference in the matter of prognosis; for, whereas secondary and tertiary nervous diseases of the ear may be favorably influenced or even cured by antisyphilitic treatment if they are taken in hand early, the prognosis in the hereditary form is unfavorable.

The term "nervous disease of the ear in syphilis" has been used designedly, as the seat of the lesion is unknown. The value of the investigations in regard to histologic changes in the labyrinths of syphilitic subjects is impaired by the fact that the etiology in these cases of alleged hereditary syphilis is doubtful (see Gradenigo [1]); and, in the second place, the changes found in secondary and tertiary syphilis—described as round-celled infiltration, calcifications, and hyperemia—are so general that nothing is gained for the pathology by the recording of such doubtful cases, which can only by much ingenuity be brought into harmony with the classic description of syphilis. There is a general tendency to ascribe syphilitic deafness to disease of the vestibule and of the first turn of the cochlea, but there is nothing to justify such an assumption, and the seat of the disease might just as well be placed in the nerve-endings or in the nerve-trunk itself.

In an interesting variety of cases the loss of hearing is due to direct lesion of the auditory nerve or of its centers by a gumma in the brain, or gummatous basal meningitis, or cerebrospinal meningitis; for the auditory nerve may be implicated in this disease as well as any of the other cranial nerves. Such a case is described by Oppenheim,[2] who in another place (p. 16) remarks that "it may eventually be possible to demonstrate the same symptoms in the auditory nerve—which, up to the present time, has been rather neglected (treated like a stepchild)—that have been accurately observed in the ocular, motor, and facial nerves." Schwartze[3] mentions a case of intracranial syphilitic paralysis of the left auditory nerve, associated with paresis of the left arm and paralysis of the tongue, but without facial paralysis; Gradenigo[4] quotes a case from Helmet of suddenly developing deafness in a young syphilitic woman, in which, at the autopsy, scattered foci of encephalitis were found, one of them at the exit of the auditory nerve-trunk.

[1] "Schwartze's Handb.," II, p. 431.
[2] "Syphil. Erkrank. des centr. Nervensystems," 1890, p. 30.
[3] "Arch. f. Ohr.," IV, p. 267 (1869). [4] "Schwartze's Handb.," II, p. 529.

X. DISEASES OF THE EYE.

1. RELATIONS BETWEEN THE EYE AND THE NOSE.

During the past few years particular attention has been directed to the relations existing between the eye and the nose, and it is being recognized more and more that pathologic conditions of the nose play an important part in the genesis of ocular diseases. Although the cases that tend to throw light on this etiologic connection are not numerous, they are all the more convincing. Seifert,[1] in a series of investigations in v. Michel's eye clinic, found nasal disease in all but 2 among 38 cases of dacryocystoblennorrhea. In another series of 48 cases the nose was regularly involved. Winckler,[2] among all the children which he examined in the course of three years in the Children's Hospital at Bremen, found the nose diseased in 50% of those suffering with scrofulous eye disease, and Ziem[3] gives it as his belief that two-thirds of all cases of ocular disease are due to disease of the nose.

It is often difficult to determine after a single examination whether or not there is any connection between the nose and the eye, as the conditions in the nose are much influenced by the presence of swelling, and the amount of mucus is variable, especially in scrofulous patients, who furnish the bulk of the material. Hence, the question whether or not the nose is diseased depends more or less on the judgment of the examiner and on his standard of regularity in structure and degree of moisture for the normal nose. Ziem appears to have the highest standard in this respect, and this may explain his large percentage of nasal disease accompanying disease of the eye, and, as will

[1] "Münch. med. Wochen.," 1898, No. 29.

[2] "Semon's Centralbl.," XII, p. 92, and "Bresgen's Sammlung," Bd. III., H. 1.

[3] "Mon. f. Ohr.," 1893, Nos. 8 and 9.

be mentioned later, his radical views in regard to interdependence between eye and nose. Thus, before admitting the integrity of the nose he subjects it to a test irrigation, as, he says, "this procedure often reveals the presence of pus which escaped the detection of anterior and posterior rhinoscopy." For my part, the finding of mucus or pus in the irrigating fluid after a nasal douche would not convince me of the existence of nasal disease unless I was able at the same time by inspection to determine the origin of the pus; if the nose is really diseased to such an extent as to be capable of affecting the eye, the diagnosis can always be made with the aid of rhinoscopy, without using a nasal douche. It is this divergence of opinion in regard to what constitutes the difference between a healthy and a diseased nose that is responsible for the different views held as to the frequency of a relationship between the nose and the eye, and for the fact that many physicians (Ziem and others) consider it a proof of etiologic connection between a nasal and an ocular disease if the ocular disease is favorably influenced by local treatment of the nose. Thus, we meet with cases of disease of the uveal tract and of visual disturbances that are ascribed to a pathologic condition of the nose, because galvanocautery or some other local interference is followed by improvement in the ocular symptoms, although no good internal evidence can be adduced to prove the connection between the two diseases.

There are three possible ways in which disease may be transmitted from the nose to the eye: through the lacrimonasal duct, through the blood and lymph streams, and by way of the nerves.

The most important rôle in the production of consecutive eye disease belongs to the lacrimonasal duct, on account of its anatomic relations to the nose. The location of its mouth in the inferior nasal meatus, below the inferior turbinate bone, close behind its anterior expanded extremity, readily explains the occurrence of disease of the tear-ducts whenever the normal drainage of the lacrimal fluid becomes obstructed, or when disease of the nose spreads to the lacrimonasal duct and to the lacrimal sac. In addition, the latter may be the means of causing disease of the conjunctiva and of the cornea by direct transmigration of pathogenic organisms from the nose to the eye.

Epiphora and blennorrhea of the lacrimal sac regularly

follow obstruction of the nasolacrimal canal. The obstruction may be due to various conditions in the nose, such as acute and chronic hypertrophies, tumors, ulcerations, and cicatricial contractions whenever they are seated in the inferior nasal meatus. Of course, the influence of a temporary disease, such as acute rhinitis, is not very great, and it is only after chronic conditions that we have lasting affections of the lacrimal sac. Among these must be mentioned particularly the hypertrophic conditions found in scrofulous children, and the polypoid hypertrophies of the lower turbinate body reaching down to the nasal floor and completely obstructing the inferior nasal meatus. Even milder grades of hypertrophy may exert a very injurious influence if the septum is deformed and its covering puckered in folds. Here belong also hypertrophic conditions of the nose due to obstruction of the nasopharynx, and we therefore find in adenoid vegetations of the pharnygeal vault one of the most fruitful sources of ocular disease. Even an atrophic rhinitis may under certain conditions lead to disease of the lacrimal sac, although the opening of the lacrimonasal duct necessarily shares in the general dilatation, for the walls of the nose, including the orifice of the lacrimonasal duct, may be entirely covered over by the closely adhering crusts of dried secretion. In this connection special mention must be made of those forms of ozena in which the lower turbinate bodies have been destroyed in consequence of caries of the bone due to the rhinitis fœtida atrophica, or genuine ozena, or to syphilitic ozena. In such cases the orifice of the lacrimonasal duct, which may be abnormally expanded as a result of atrophy of the mucous membrane or of cicatricial changes, opens directly into the nasal cavity, so that the crusts which cover the nasal walls may completely occlude it, an event which can not take place as long as the inferior turbinate bone is present and affords a certain protection. Thus, we see some of the most obstinate cases of blennorrhea of the lacrimal sac in hereditary syphilitic ozena, in which, particularly in the case of young children, the turbinate bones and the septum are destroyed and the entire nasal cavity is completely filled with hard, stinking crusts, which can be removed only with great difficulty by means of the douche and a cotton-carrier. Their rapid recurrence can, at best, only be delayed by the most conscientious regularity in treat-

ment, so that we can readily understand the frequent relapses and the chronic course of the ocular complication. Tumors rarely lead to obstruction of the inferior nasal meatus in their early stages, as they usually spring from the region of the ethmoid bone. The same is true of nasal polypi, as they rarely occur in the anterior half of the lower turbinate body, and can not, therefore, affect the tear-ducts by direct obstruction of the orifice; they do not become important until they have grown so large and so numerous as to fill every part of the nasal cavity.

Lastly, we have ulcerative processes and granulations, such as occur in tuberculosis, lupus, syphilis, rhinoscleroma, leprosy, glanders, etc. These may lead to stenosis or occlusion of the canal, even after they have healed, by reason of the cicatricial contractions and adhesions which remain.

The effects of the nasal disease are not always limited to occlusion of the lacrimonasal duct; the infection may spread through the canal of the eye itself and lead to inflammations of the lacrimal sac, to conjunctivitis, and to keratitis. It is in this way that we explain the occurrence of eczematous keratitis and conjunctivitis in connection with eczema of the vestibule and chronic hypertrophic rhinitis in scrofulous individuals, as demonstrated by Knies in 90% of cases occurring in children.[1] It is interesting to note that Seifert[2] found rhinitis fœtida atrophica in the great majority of all cases of spreading ulcer of the cornea, so that he was led to infer the extension of an infection of the cornea from the nose. Buck [3] mentions corneal ulcers following ozena. According to Fuchs,[4] ozena is a frequent complication of trachoma; and Klunzinger, Ziem, Gerber, and Kuhnt assume a connection between trachoma and disease of the nose, on the ground that the "granulosis of the nose" may set up a secondary granulation in the lacrimal apparatus and on the palpebral conjunctiva—a view which is not confirmed by other authors. Although Löwenberg's ozena bacillus has been found in the conjunctival sac by Terson and Gabrielidès, [5] and although Abel [6] also found his *bacillus mucosus*

[1] Knies, "Die Beziehungen des Sehorgans und seiner Erkrankung," p. 285.

[2] "Münch. med. Wochen.," 1898, No. 29.

[3] Ref. "Semon's Centralbl.," XI, p. 217.

[4] "Lehrb. der Augenheilk.," p. 570.

[5] "Arch. d'ophthalm.," XIV, p. 488, quoted from Schmidt-Rimpler. "Nothnagel's Handb.," XXI, p. 430.

[6] "Zeitschr. f. Hygiene," Bd. XXI, H. 1.

ozænæ, no local lesion directly due to the micro-organisms could be demonstrated in the eye in either case.

Despite the fact that it is generally assumed that diseases are transmitted through the lacrimal duct only from the nose to the eye, and that transmission in the other direction is not considered important, this method does, nevertheless, appear to play some part in gonorrheal infection of the eyes, as Miller [1] repeatedly found the nasal mucous membrane diseased in blennorrhea neonatorum, and was able to demonstrate the presence of gonococci.

Transmission of tuberculosis of the nose to the lacrimal sac has been observed (Wagonmann-Fuchs [2]), and lupus also may spread to the eye.

Batut [3] reports two cases of diphtheric disease of the nose and eye without bacteriologic findings.

It is well known that there is an intimate relationship existing between the *vascular system* of the nose and that of the eye. Arterial anastomosis between the nose and the eye is effected by means of the ethmoid arteries, by branches of the ophthalmic, and by a collateral trunk along the lacrimonasal duct, which joins the angular, the ophthalmic, and a branch of the infra-orbital artery—(Zuckerkandl). In the same way a communication is established by means of a network of veins between the lacrimal plexus and the veins of the nose, the orbit, and the face ; besides, there are larger venous trunks running from the nose to the cranial and orbital cavities—the ethmoid veins. Ziem lays the greatest stress on the connections between both the arterial and the venous systems in the etiology of ocular disease accompanying disturbances of the nasal circulation, which occur in acute and chronic inflammations and in the passive hyperemias that are so common in the nose. But as these disturbances are followed by disease of the eye only in comparatively rare instances, Winckler believes that the cause is to be sought in individual anomalies in the anastomoses.[4] Conjunctivitis as well as blepharitis and epiphora are frequently observed to follow circulatory disturbances of this kind occuring in acute and chronic hypertrophic conditions, and in almost every variety of nasal stenosis, but the doc-

[1] Stōrk, "Nothnagel's Handb.," XIII, 1. Th., 1. Abth., p. 86 (note).
[2] "Lehrb. der Augenheilk.," p. 570.
[3] "Ann. des mal. de l'oreille," 1893, p. 114.
[4] E. Winckler, "Bresgen's Sammlung," III, H. 1.

trine of Ziem [1] that diseases of the uveal tract often originate in this way has not found many adherents.

The following cases are probably to be ascribed to circulatory disturbances: Straub [2] reports a case in which there were attacks of pain and congestion in both eyes, lasting from two to six days, accompanied by epiphora and protophobia; he assumes a vasomotor neurosis originating in the nose, as removal of a crista septi and cauterization of the hypertrophic turbinate bodies was "followed by almost complete cure." Dunn [3] saw a case of recurring edema of the upper eyelid which disappeared after removal of the anterior extremities of both middle turbinate bones, which were the seat of polypi.

The fifth nerve supplies a part of the nose through a branch of its first division; the innervation of the septum, the vestibule, and the external skin of the nose being effected by the external and internal branches of the nasal nerve.

This nervous connection explains the reflex sensations in the nose—tickling and sneezing—which follow irritation of the ciliary nerve when the eye is suddenly subjected to a strong light, as, for instance, when we look into the sun, or in inflammatory disease of the eye. On the other hand, irritation of the ocular nerves by way of the branches of the fifth which we have just described in disease of the nose is much more common. Its simplest expression is seen in the redness of the conjunctiva and the lids and in the increased flow of tears which follow the slightest local interference in the nose, even the mere touching of the corresponding side of the nose with a probe. In this category we may include a ciliary neurosis described by Seifert,[4] due to synechiæ, after extensive cauterizations in the interior of the nose.

Quite a number of other ocular affections have been ascribed to primary nasal disease, without, however, sufficient proof of the etiology and the manner of the reflex influence being forthcoming. Thus, for instance, Laurens [5] divides reflex disturbances of the eye into those which

[1] Compare "Mon. f. Ohr.," 1893, p. 262.

[2] "Nederl. Tijdschr. v. Geneesk.," 1896; see "Semon's Centralbl.," XII, p. 425.

[3] See "Semon's Centralbl.," IX, p. 371.

[4] *Loc. cit.*

[5] "Ann. d'ocul.," April, 1896; see "Semon's Centralbl.," XII, p. 426.

"affect the general or special sensibility of the eye (neuralgias, photophobia, amblyopia); reflex disturbances of the motility (blepharospasm, mydriasis, strabismus, asthenopia); and, finally, nutritive and vasomotor disturbances in the coverings of the eye (conjunctivitis, iritis, glaucoma, exophthalmos)." The danger of exaggeration in artificially constructing such relations can not well be emphasized too strongly. The reflex connection between the eye and the nose, through the agency of the trigeminus, is much less extensive than might be supposed from the statements of many authors (Fortunati, for instance), who would ascribe to the second division of the trigeminus the power of producing reflex disturbances in the eye through its nasal ramifications, although Ziem goes to the opposite extreme and attributes the origin of secondary diseases of the eye principally to the agency of the vascular system. In proof of the reflex influence of nasal diseases cases are reported by Knies,[1] Schmidt-Rimpler,[2] Lieven,[3] and E. Winckler,[4] but I shall not repeat them here, as they neither prove nor explain anything.[5] I may, however, mention a few rather spurious examples taken from the latest literature. Laurens [6] observed a case of blepharospasm which disappeared after obstruction of the nose due to hypertrophy of the mucous membrane and synechiæ had been corrected. He also reports seeing a six-year-old girl with left converging strabismus, which disappeared after an operation for adenoid growths. On the other hand, Baumgarten [7] considers strabismus, which he observed twice in hypertrophy of the pharyngeal tonsils, an accidental complication, as it was not influenced by operation. Schloss and Myles [8] report several cases of asthenopia which subsided after removal of hypertrophied turbinate bodies, the removal of a spine on the septum, and, in some cases, of tumors. Myles believes that hypertrophies of the tissues, by pressure on the nerves, provoke ocular symptoms, but this supposed connection with the nose was not proved to exist in all the cases in which a nasal opera-

[1] *Loc. cit.* [2] *Loc. cit.* [3] *Loc. cit.* [4] *Loc. cit.*

[5] See reports in "Semon's Centralbl."

[6] "Presse méd.," 1896, Jan. 22; see "Semon's Centralbl.," XII, p. 425.

[7] "Neurosen und Reflexneurosen des Nasenrachenraumes," "Volkmann's klin. Vortr.," N. F., No. 44.

[8] "Pacific Med. Jour.," 1894, and "N. Y. Med. Record," 1894; see "Semon's Centralbl.," XI, pp. 280 and 281.

tion was performed. Bernstein [1] speaks of improvement in errors of refraction after removal of nasal hypertrophies. According to Knies,[2] operative interference on the nasal membrane is rarely followed by visual disturbances consisting in concentric narrowing of the visual field with or without disturbance of the central sight and of the color-sense. Fortunati [3] assumes a nasal origin for two cases of neurokeratitis in which ulceration and perforation of the cornea, with prolapse of the iris, occurred after a long-continued obstruction of the nose. Winckler [4] reports a case of retrobulbar optic neuritis with serous tenonitis which was treated for six weeks without benefit, and was finally cured within a month after removal of papillomata on the turbinate bodies. Pupillary changes—mydriasis and myosis—have also been described as due to nasal irritation, as the snuffing-up of cold water into the nose (Ostmann [5]).

Inflammatory disease of the accessory cavities is always accompanied by hypertrophic and polypoid alterations in the interior of the nose, which may in turn lead to the disturbances we have just described. After Ziem, the pioneer in this field, Kuhnt[6] deserves the credit of describing in a comprehensive work the dependence of ocular complications on diseases of the accessory cavities, thereby awakening the interest of other investigators in many questions hitherto much neglected. The lines he laid down were followed among others by Schmidt-Rimpler, who has produced the latest work on this subject, while the same questions have also been extensively dealt with by Grünwald.[7] The latter has collected a large number of cases.

The anatomic position of the accessory nasal cavities is such that a morbid process originating within them is easily transmitted to the orbits. The lateral wall of the ethmoid cells, consisting principally of the os planum (lamina papyracea), and completed at the anterior and posterior ethmoid walls by the juxtaposition of the lacrimal bone and the orbital processes of the palatal bone, also forms the

[1] "Med. News," July 22, 1893; see "Semon's Centralbl.," X, p. 386.
[2] *Loc. cit.*, p. 288.
[3] "Arch. d'otol.," 1896, No. 2; see "Semon's Centralbl.," XIII, p. 330.
[4] "Semon's Centralbl.," XII, p. 92. [5] "Arch f. Ophthalm.," 43.
[6] "Ueber die entzündlichen Erkrankungen der Stirnhöhle und ihre Folgezustände," 1895.
[7] "Die Lehre von den Naseneiterungen," 2d ed.; 1896, p. 122.

median wall of the orbits; immediately above it lies the frontal sinus, and beneath its floor the antrum of Highmore, so that all these cavities have at least one wall in common with the orbit. On the other hand, the sphenoid sinus, the last of the series of cavities which make up the accessory pneumatic system of the nose, does not possess a very intimate relation with the eye, except that the robust layer of bone which forms its roof lies in apposition with the interior of the cranium, and in rare cases endocranial perforations may be produced and lead to direct injury of the optic nerve, but accompanied, usually, by other anatomic complications. Berger[1] remarks that even a simple inflammation of the sphenoid sinus may lead to retrobulbar optic neuritis when the opticosphenoid wall is unusually thin or is traversed by fissures.

Although from an anatomic point of view a pathologic relation may exist between these accessory cavities and the eye, clinical experience teaches that only certain diseases appear to possess a tendency to spread to the orbital cavity, depending on the seat and the nature of the particular disease. The commonest way in which sequels occur in the eye is when in acute or chronic inflammation of the accessory cavities a serous or purulent exudate is retained, and thus produces bulging of the cavity walls. The likelihood of pus being retained in an accessory cavity depends on its relative position and on the size of the openings by which it normally communicates with the interior of the nose; that is to say, the more imperfect the drainage, the greater the danger of retention. Thus, in the antrum of Highmore and frontal sinus the nasal openings are so unfavorably situated that even a slight alteration in the neighborhood of the opening is capable of producing retention. While in the antrum the median wall, which lies toward the nasal cavity, the exterior wall (canine fossa), or the palatal bone is more likely to bulge than the roof of the cavity, directed toward the orbit, yet in the frontal sinus it is the orbital wall, which corresponds to the floor of the cavity, that is more liable, on account of its extreme tenuity, to break down under the weight of the accumulated secretion than is the more robust anterior wall. Bulging of the lateral nasal wall due to empyema of the antrum may

[1] "Soc. franç. ophth.," May, 1894; see "Semon's Centralbl.," XI, p. 573.

in rare cases produce compression of the lacrimal duct. Such an event is much less frequent, however, than displacement of the globe outward and downward by bulging of the orbital wall of the frontal fossa and the resulting disturbances in the mobility, function, and drainage of the eyeball. The protrusion of the eyeball is usually preceded by edema at the upper inner angle of the orbit, at which point empyema of the sinus sometimes ruptures into the orbit and leads to orbital phlegmon. From the tenuity of the os planum, which separates the ethmoid cells from the orbit, we should expect to see empyema of these cells followed by disturbances in the eye. This is not the case, however, as there is little tendency to retention of the pus, because the outlets of the cells toward the nose are short and spacious, and the walls of the cells are so thin on either side that perforation into the nose easily takes place.

These remarks do not by any means exhaust the subject of the relations existing between the eye and disease of the accessory cavities; there is a host of inflammatory and functional disturbances which are said to accompany and to be dependent on inflammatory conditions of these cavities. Hyperemic and catarrhal conditions of the conjunctiva and cornea, and diseases of the uveal tract, by subsiding after the recognition and treatment of suppuration in the accessory cavities, appear to indicate a mutual relationship, although Kuhnt[1] justly observes that the removal of inflammatory conditions in the accessory cavities plays only a secondary part in the treatment of ocular diseases, and merely assists and reinforces the general treatment; he does not believe that the ocular disease can be cured in this way without careful local treatment of the eye.

When we come to functional disturbances, we have hyperemia and venous stasis of the papilla, and peripapillary opacity of the retina in suppuration of the frontal sinus of the same side, which, according to Kuhnt,[2] always disappears after removal of pus, thus indicating a connection with the disease of the accessory cavity. When restrictions in the field of vision occur, they are usually bilateral, although more marked on the affected side. They are usually accompanied with weakness of the internal muscles (Kuhnt). For the sake of completeness we

[1] *Loc. cit.*, p. 112. [2] *Loc. cit.*, p. 121.

may mention that Kuhnt does not absolutely deny Ziem's statement that cataract may be produced by suppuration in an accessory cavity. Careful investigation is urgently needed before the dependence of all these conditions on suppurations in the accessory cavities can be accepted as proved. But meanwhile the meager clinical material that has been contributed by reliable investigators is not to be disregarded, even if the explanation offered is not always quite satisfactory. Kuhnt's theory that absorption of purulent or fetid masses from the diseased cavities plays the principal part in the etiology of functional disturbances of the eye, and not the vascular system, as Ziem contends, deserves attention. The effect on the nervous system of this absorption varies with the individual, and may be responsible for a rapid tiring or even a kind of obtuseness in the optic tract and in the nerve-endings of the retina.

Noninflammatory diseases in the accessory cavities, such as malignant tumors, carcinomata, and sarcomata, may spread to the orbits and lead to appearances identical with those of orbital tumors. Photiades[1] reports a reflex mydriasis due to endolaryngeal interference for the removal of laryngeal polyps.

2. RELATIONS BETWEEN THE EYES AND THE EARS.[2]

The eye may be influenced by the ear in various ways, and may furnish valuable diagnostic points to the otologist; while, on the other hand, diseases of the eye do not involve the ear, if we except a few scattered observations relating to the impairment of the power of hearing or to the production of tinnitus aurium by sudden flashes of light, or such cases as Stevens',[3] in which division of a slightly insufficient internal rectus was followed by the disappearance of tinnitus aurium, or where, after iridectomy for glaucoma and optic iridectomy in leukoma of the cornea an improve-

[1] "Semon's Centralbl.," pp. 277, 278.

[2] For extensive report of cases see Ostmann, "Arch. f. Ophthal," 43, p. 22; Schmidt-Rimpler, Nothnagel's "Spec. Path. u. Therap.," vol. XXI, p. 435; Knies, "Beziehungen des Sehorgans und seiner Erkrankung," etc., 1893, p. 289.

[3] Stevens, "Arch. f. Ohr.," XIX, p. 75.

ment in hearing was noted,[1] or where, as reported by Wolf,[2] subjective aural sensations occurred during attacks of glaucoma.

Reflex irritation of the eye originating in the ear plays an important part in these relations, while direct injury of the optic nerves depends rather on endocranial sequels of optic disease than on disease of the ear itself. Reflex irritation may give rise to disturbances in the function of the eye muscles, as the vestibular and cochlear branches of the auditory nerve are in close relation with the optic pathway. There is no doubt that the vestibular nerve may exert an influence on the coordinating center that presides over the action of the ocular muscles, and that irritation of the nerve itself or of its endings in the ampulla and in the membranous semicircular canals may produce motor disturbances in the domain of the oculomotor, the abducens, and the trochlear nerves, manifesting themselves in nystagmus, ocular palsy, and disturbances in the pupillary reaction. This has been proved by numerous physiologic experiments on disturbances of equilibrium following injury of the labyrinth, and particularly by Stein,[3] who was perhaps somewhat hasty in applying unfinished theories to practical diagnosis. This reflex connection has been utilized in the diagnosis of aural vertigo. For the transmission of reflex irritation from the ear to the eye by means of the vestibular nerve we possess some anatomic basis, but for the connection between the cochlear nerve and the ocular nerve the anatomic basis is not equally clear, and rests solely on the occurrence of aural hallucinations, as described by Bleuler, Lehmann,[4] and Urbantschitsch.

We have, however, in Held's investigations an important contribution to the physiology and pathology of the nervous system, which may eventually lead to the overthrow of the vague theories at present prevailing concerning the reflex connection between the ear and the eye, and furnish a positive proof that auditory stimuli are capable of affecting the movements of the muscles. Held's[5] investigations resulted in the demonstration of a reflex arc by which audi-

[1] Knies, *loc. cit.*, p. 291.

[2] "Arch. f. Augen u. Ohr.," IV.

[3] Arb. a. d. Bazanow'schen Klinik I, 1, Moscow, 1897; "Zeitschr. f. Ohr.," vol. XXVII.

[4] "Zwangsmässige Lichtempfindung durch Schall und verwandte Erscheinungen auf dem Gebiete der Sinnesnerven."

[5] "Arch. f. Anat. und Entwickelungsgesch.," 1893, p. 201.

tory stimuli may be transmitted to the motor apparatus of the eye, for he proved that auditory stimuli can be communicated to the oculomotor, trochlear, and abducens nerves by way of the reflex arc common to the optic and auditory nerves, having its origin in the anterior corpora quadrigemina. This same reflex arc also includes other paths by which auditory stimuli may reach the nucleus of the facial nerve and the formatio reticularis, and can probably be transmitted from these to the respiratory, vasomotor, and other centers.

The reflex movements which follow auditory stimuli, and consist in turning the eyes or the head toward the side from which the sound proceeds, may be explained in the same way; they suggest the possibility of pathologic processes in the ocular muscles manifesting themselves as atactic movements, being produced by improper or irregular irritation of the cochlear nerve.

If auditory stimuli are capable of producing coordinated movements of the eyes by means of this reflex arc when the hearing is normal, it is conceivable that when in disease of the ear the sound is not heard with equal intensity on both sides, the sound waves, being perceived in a different way on the two sides, may possibly produce a different reflex irritation on the two optic tracts.

If the coordinating center for a properly regulated movement of the eye receives an impulse of normal strength from one cochlear nerve and a weaker impulse, or none at all, from the other, the equilibrium in the coordinated muscular movement may be disturbed, and atactic movements of the optic muscles are produced. In this way we may perhaps explain cases like Bürkner's,[1] in which the effort of the right ear, which was the seat of a suppuration, to catch the sound during the functional test was followed by nystagmus. The effect is always bilateral, because of the decussation of the deep roots of the ocular nerves; unilateral reflex disturbance of the eye through the ear is impossible.

The ocular phenomena that follow increased pressure in the middle ear are due to the pressure changes communicated to the labyrinthine fluid by the simultaneous pressure on the fenestræ, and it is probable that the reflex irritation follows the same paths of the vestibular and coch-

[1] "Arch f. Ohr.," XVII, p. 185.

lear nerves that we have just described. Lucae [1] was able, by raising the pressure in the middle ear through the external meatus in a case of perforated ear-drums, to produce vertigo, which was proved to be optic in character by the fact that it immediately disappeared when the eyes were closed. As in Lucae's cases crossed double images were produced, he argues "that the increased pressure led to irritation of the abducens nerve."

I have given this short description of the physiologic possibility of reflex ocular movements being produced by irritation in the ear so as to throw some light on the clinical cases which have been described as belonging to this category.

Nystagmus has been said to follow irrigation of the external meatus, and to occur in cerumen concretions, in purulent otitis media, and after extraction of polyps from the middle ear. In those cases where the reflex is produced by local influences in the external meatus and on the drum membrane, in irritation and in accumulations of cerumen, reflex irritation must be explained by pressure changes in the labyrinthine fluid, as in the case described by Lucae. To what extent the trigeminus may be concerned in reflex connections between the ears and the eyes is not definitely known, although reflex irritation of the ocular muscles through this nerve seems possible in view of the connections which are known to exist anatomically. That the trigeminus may be concerned in the reflex irritation appears to be indicated by the fact that the temperature of the fluid used in irrigation has some effect on the production of reflex ocular movements, as very cold or very hot water appears to favor their occurrence. Lucae's observation that the reflex irritation which occurred when the ear-drum was perforated was absent when that membrane was intact, can not be utilized to determine whether the irritation affects the mucous membrane of the middle ear directly or not, as individual peculiarities appear to come into play that can not be overlooked.

It appears to be proved by experience that disturbances of coordination may be produced in the movements of the eye muscles by pressure changes in the middle ear, such as are frequently observed in catheterization. For similar

[1] "Arch. f. Ohr.," XVII, p. 237.

disturbances in the course of an acute or chronic purulent otitis media, however, the proof is not so clear, as there is no anatomic basis for a direct reflex irritation on the ocular nerves by inflammatory conditions in the middle ear, unless it be by means of the tympanic plexus, and this is exceedingly doubtful. The occurrence of nystagmus or other motor disturbances in the eye in cases of purulent otitis media probably depends on labyrinthine or intracranial complications of the ear affection. Ostmann [1] says that "ocular symptoms occurring in the course of acute purulent otitis media must be regarded as due to tonic spasm within the labyrinth or to an intracranial sequela." Jansen [2] considers nystagmus a somewhat vague symptom, most probably to be referred to an affection of the labyrinth, and, in the absence of such an affection and of leptomeningitis, he attributes to it a certain significance for the diagnosis of sinus phlebitis or periphlebitis in the neighborhood of the temporal bone. It may occur in extradural abscesses as the result of pressure on the occipital lobe and on the cortical centers for the ocular movements which it contains. He describes nystagmus as bilateral and synchronous, as a horizontal or a rotatory and vibratory movement, usually short and sharp, sometimes slow and more extensive, occurring during fixation of the eyeball. It appears principally when the glance is directed away from the affected ear, sometimes as soon as the median line is passed, and increases as the eye is moved farther away, whereas when the eyeball is rotated toward the affected ear it remains in a state of complete rest, or at most indulges in a few interrupted movements.

Our knowledge of pupillary anomalies during purulent otitis media is very meager. The phenomenon has been reported by Schwartze [3] and by Moos.

While reflex irritation of the trochlear and abducens nerves by way of the trochlear nerve is well known to occur physiologically after an auditory impression, and probably occurs also under pathologic conditions, paralysis of these nerves in diseases of the middle ear and of the labyrinth must always be referred (as pointed out by Habermann [4]) to intracranial complications, and in the

[1] "Arch. f. Ophthal.," p. 13. [2] "Arch. f. Ohr.," XXXVI.
[3] "Arch. f. Ohr.," XVI, p. 263.
[4] "Verhandl. der D. otol. Gesellsch.," 1898, p. 98.

absence of any conspicuous alterations at the autopsy it is to be explained by the existence of a serous meningitis or an inflammation of the pia too slight to attract attention.

Such palsies of the ocular muscles have frequently been observed after intracranial complications of an ear affection, and in the latest literature [1] on the sequels of diseases of the ear we find them described as depending on the mode of extension of the disease to the sinuses, the serous membranes, and the brain-substance itself.

Another important symptom that accompanies these conditions is papillitis of the optic nerve. When it occurs in a purulent otitis media, if there is a suspicion of intracranial complication it is of vital significance, in spite of Jansen's [2] statement that "it appears to occur in rare cases, even in uncomplicated empyema of the mastoid process or in otitis media, through the agency of the carotid plexus," and should always be regarded as a proof that the inflammation has spread to the interior of the cranium. When chronic purulent otitis media is associated with congestive papillitis and cranial symptoms, it becomes very important to determine whether the two diseases have anything to do with each other or not. In tuberculous patients with chronic middle-ear disease it must always be borne in mind that the ocular and cranial symptoms, which may appear to simulate otitic meningitis or an extradural or cerebral abscess, may have their origin in a tubercular meningitis or in cerebral tuberculosis and be in no way dependent on the aural affection.

Sensory disturbances in the eye may be secondary to earache transmitted from the tympanic plexus by way of the trifacial nerve; they manifest themselves in the eye as pain, increased lacrimation, and injection of the conjunctival vessels.

Urbantschitsch's [3] statement that visual acuity may be affected by aural disease is not borne out by the results of Ostmann's [4] investigations. The mutual influence of auditory and ocular impressions, which are described by Bleuler and Hoffman [5] under the names of "Gehörsphotismen"

[1] Jansen, "Arch. f. Ohr.," XXXVI; Hessler, "Otogene Pyämie"; Körner, "Die otitischen Erkrankungen," etc.

[2] "Arch. f. Ohr.," XXXVI.

[3] "Pflüger's Arch.," XXX, p. 129.

[4] "Arch. f Ophthal.," p, 43.

[5] Quoted from Urbantschitsch, "Schwartze's Handb.," I, p. 451.

and "Lichtphotismen" (aural and ocular hallucinations) are as yet of no clinical value.

The occurrence of blepharospasm with spasm of the stapedius muscle is to be explained as a reflex irritation due to the fact that both the stapedius muscle [1] and the orbicularis palpebrarum derive their innervation from the facial nerve.

[1] Gottstein, "Arch. f. Ohr.," XVI, p. 61.

XI. INTOXICATIONS.

The upper air-passages are very much exposed to local injury during intoxications, both when the poison is contained in the air and thus comes into immediate contact with the mucous membrane of the nose, the pharnyx, and larynx, and when it is ingested in the form of a fluid or solid, and during its passage through the pharynx inflicts direct injury on that structure and on the upper margin of the larynx. From this point the poison may make its way into the interior of the larynx and set up an extensive morbid process. The ear escapes, as a rule, unless the tubes are involved in hypertrophic conditions of the post-nasal space; hence, the number of substances capable of exerting any influence on the ear when taken by the mouth is very limited.

The most frequent symptoms produced by the great majority of organic and inorganic chemic bodies by direct irritation of the mucous membrane of the upper air-passages are hyperemia and sensory irritative phenomena, such as sneezing and coughing.

Their recognition presents no difficulty, as the cause of the intoxication can usually be ascertained, and the clinical picture presents no special characteristics for the individual kinds of intoxications, so that it is not worth while to enumerate all the various acids, alkalies, ethereal oils, etc., in this place.

Another group of symptoms which it is customary to refer to the action of various poisons can not be accepted as toxic phenomena without a reservation. Among these we have aphonia, hoarseness, and tinnitus aurium. The former is due to "adynamia, when the constitutional effect of the poison has so debilitated the entire organism that the phonetic function shares in the impairment of all the other functions, especially those of the central nervous system" (Stuffer [1] on "Toxic Aphonia"). In the literature of alkaloid poisoning we find in particular nervous disturbances

[1] "Arch. f. Laryng.," vol. VI.

of speech mentioned along with these adynamic phenomena, so that mistakes are very apt to be made in interpreting the findings.

The same applies to the auditory disturbances, which are usually given as tinnitus aurium. When we consider the manifold causes that may give rise to this phenomenon; how frequently it is due to circulatory disturbances, which play so important a rôle among the toxic effects of many poisons; and that tinnitus aurium, and even hallucinations, often occur after the exhibition of stimulant remedies, we realize how easy it is to refer symptoms which really originate outside of the ear to a direct toxic effect of the poison on the ear itself.

We shall, therefore, mention only those substances which produce marked clinical disturbances clearly due to the constitutional effect of the poison, leaving out all the symptoms of a vague and indefinite character.

Acids and alkalies exert a direct caustic effect on the mucous membranes that manifests itself in various ways. The effect of acids is chiefly that of a cauterizing agent, causing constriction of the tissues and the formation of crusts,—that is to say, the effect is more superficial and is localized in the region where it is applied,—whereas alkalies tend to dissolve the tissues and to produce deep destruction involving the entire surface of the mucous membrane and not confined to the area of contact. In both cases the affected part becomes surrounded by an area of marked inflammation and swelling. As the ingestion of liquid poisons is always accompanied with the cauterization of the pharynx and of the entrance to the larynx, —that is, of the epiglottis and the aryepiglottic folds,—the edema which follows may be very extensive, and the patient's life is endangered more by stenosis of the larynx than by the toxic effect of the substance itself.

The manner of healing and cicatrization similarly varies in accordance with this difference in the effects of acids and alkalies; in the former the resulting scars are smooth and superficial, while in the latter we have deep cicatricial contractions, and particularly the formation of cicatricial adhesions uniting the upper margin of the larynx with the deeper portions of the pharyngeal wall.

The commonest examples of these two kinds of intoxication are poisoning with sulphuric, hydrochloric, and nitric

acids on the one hand, and poisoning with potassium or sodium hydrate and ammonia on the other hand. That chlorid of zinc is capable of producing the same kind of destruction of the mucous membranes as an acid is shown by a case of v. Jaksch's [1], in which, after the drinking of a solution of chlorid of zinc and hydrochloric acid, such as is used in soldering (68 gm. of zinc chlorid and 3.5 gm. of hydrochloric acid to 100 c.c.), laryngeal stenosis occurred which necessitated tracheotomy. Among the intoxications by inorganic acids we must mention particularly chromic acid poisoning, as this substance is a favorite cauterizing agent in rhinologic practice. Acute chromic acid poisoning may follow the use of only a few centigrams, as in cauterization of the pharynx, and leads to a general intoxication in addition to the local symptoms; while, on the other hand, the chronic form of poisoning, which occasionally occurs in employees in chromic acid factories, produces deep-seated alterations of the mucous membranes. The inhalation of chromic acid in the form of dust at first leads to an inflammation of the nasal mucous membrane, which is soon followed by arrosions on the septum and on the anterior extremities of the turbinate bones, constantly accompanied by epistaxis. Ulceration also takes place in regions to which the particles of dust may be carried by the inspiratory air: that is, the tonsils, the uvula, and the posterior pharyngeal wall. According to Seifert,[2] purulent inflammation of the tympanic cavity may also occur by extension through the Eustachian tubes.

The internal administration of the iodids, especially potassium iodid, is sometimes followed by alarming symptoms in the upper air-passages. It is well known that the exhibition of iodin is always accompanied by a slight swelling, redness, and desquamation of the mucous membranes, manifesting themselves in more or less marked coryza, lacrimation, pharyngitis, and laryngitis. But, in addition, the literature contains a number of intoxications following the use of potassium iodid which led to alarming symptoms, and in a few cases even necessitated tracheotomy. The symptom referred to is edema of the larynx. It has been observed in every part of the larynx—on the lateral wall, about the entrance, on one side of the larynx only, or on

[1] Nothnagel's "Spec. Path. u. Therap.," vol. I.

[2] "Die Gewerbekrankheiten der Nase," etc., Fischer, Jena, 1895.

both sides in the subglottic region. Our knowledge of its cause and of its mode of origin is very meager. The intoxication does not appear necessarily to follow large doses of the drug, as cases have been reported in which a short course of treatment with small doses produced an intoxication (Rosenberg). In two cases reported by Schmiegelow [1] in which tracheotomy had to be performed, edema occurred after the administration of three teaspoonfuls of a 5% solution taken morning and evening in one case, and in the other case after only three tablespoonfuls of the same solution had been taken three times a day for several days. The cases in which the intoxication occurred after withdrawal of the drug (Heymann), or after it had been used for several weeks, must be considered exceptional, for we know that, as a rule, the mucous membrane becomes accustomed to the drug after a few days of catarrh; and, even in those cases in which edema of the larynx had occurred after a few days' use, the drug was subsequently very well borne when it was given in more conservative doses. The manner in which the intoxication occurs is as little known as its cause; it is remarkable how seldom grave toxic appearances are seen when we consider the enormous number of cases which are constantly treated with potassium iodid. Rosenberg believes that the occurrence of intoxication depends on the presence of glands; Avellis, arguing from an interesting case of unilateral paralysis of the recurrent nerve in which the administration of potassium iodid was followed by edema of the larynx on the unaffected half of the larynx only, suggests that iodid poisoning takes place by way of the nerves, like the angioneuritic edema of Strübing; while G. Lewin, in the face of antagonistic observations reported by Rosenberg and others, assumes that iodid edema depends on syphilitic disease, on the ground that a syphilitic ulcer reacts more intensely to iodin.

The aural symptoms observed after the use of potassium iodid consist in tinnitus aurium associated with difficult and double hearing. The first two phenomena occur in association with catarrh of the pharyngeal mucous membrane, which has led to acute catarrh of the tubes and its consequences; but it seems to me we may also assume that the

[1] "Arch. f. Laryng.," vol. I.

iodin may exert a direct influence on the mucous membrane of the middle ear in the form of swelling and exudation. With regard to the remarkable phenomenon of double hearing, Moos reports a case in which, after the potassium iodid had been taken for six weeks, there followed, in addition to the iodin eruption and coryza, a peculiar affection of the left ear, so that the notes from d to g were heard double, each perception being separated by a short interval. I myself once observed, after the use of potassium iodid (5%), a case of double hearing for all the sounds of ordinary conversation which only subsided several weeks after the withdrawal of the drug. In the treatment of iodin intoxication the preparations of belladonna and sodium carbonate have been recommended.

Arsenic is used in many of the arts, and leads to diseases in the nose, in the postnasal space,[1] and in the larynx, while the ear is not affected, if we except ulcers in the external auditory meatus. When arsenic is taken internally, especially in the case of arsenic eaters, aphonia and, according to Seligmüller,[2] paralysis of the vocal cords are observed. Unfortunately, these statements lack the support of actual observation. The only well-reported case of paralysis of the left recurrent nerve said to be due to arsenic is contributed by P. Heymann,[3] but unfortunately it admits of more than one interpretation, as the patient was exposed to the fumes of cyanid gas as well as to arsenic, and the paralysis may therefore have been due to the effect of that substance.

The catarrh of the nose and of the post-nasal space, which, according to Seifert, has been observed by many authors, is to be referred to the inhalation of dust particles containing arsenic, especially the color known as "Schweinfurt green." The excoriations and ulcers which result affect particularly the septum, and often lead to perforation of the cartilaginous portion. Toeplitz found perforations of this kind in 19 out of 31 employees in a chemic factory where Schweinfurt green was made; in other words, in 61.3%.

Paralysis of the laryngeal muscles occurs in chronic lead-poisoning, the substances being usually hydroxid of lead, lead oxid, and red peroxid of lead (Mennige). To

[1] Compare Seifert, "Die Gewerbekrankheiten," etc.
[2] "Die Krankheiten der Nerven." [3] "Arch. f. Laryng.," vol. IV.

judge by the numerous descriptions, the clinical picture varies a good deal, and there is no typical form for the paralysis. Seifert, Schech, Krause, and P. Heymann have described paralysis of various muscles, including the adductors and abductors. We have individual palsies of the cricoarytenoideus lateralis, and of the interarytenoideus; unilateral and bilateral paralysis of the posticus and of the recurrent laryngeal nerve; and we can not agree with M. Mackenzie when he says that the "adductors only are implicated, just as in cases of systemic lead-poisoning the extensors are exclusively affected." Krause mentions a peculiar form of phonatory disturbance, an intention tremor, as a result of lead-poisoning. The prognosis is favorable. The three cases observed by Heymann all ended in recovery after the customary treatment for lead-poisoning.

In the ear the effects of lead-poisoning have been described as tinnitus aurium and a gradual deterioration in the power of hearing. Wolf [1] reports several cases in which the degree and kind of deafness were variable, so that the functional test sometimes appeared to locate the seat of the disease in the middle ear, at others in the internal ear. In one of Wolf's cases, in which the onset was acute, he assumes an acute exudation into the cochlea which underwent absorption after treatment, and thus allowed the function to be restored. We may assume a neuritis of the auditory nerve analogous to that which occurs in other cranial nerves, such as the vagus and the optic nerve, without, however, neglecting the etiologic importance of the arteriosclerotic condition of the vessels which accompanies the intoxication. [2]

Mercurial poisoning is well known under the name of "ptyalism," and affects the mucous membrane of the mouth and pharynx, while, as far as we know, the nose and larynx escape. Von Jaksch [3] describes a case of acute sublimate poisoning following the ingestion of a teaspoonful of the drug. The mucous membrane of the uvula, the pharynx, the epiglottis, and the aryepiglottic folds were greatly swollen and covered with a whitish exudate, while the vocal cords were only slightly inflamed. On the fourteenth day after the accident ulcers were found in these regions, the swelling had subsided, and at the autopsy, which was held on

[1] "Verhandl. der D. otol. Gesellsch.," 1895.
[2] Compare Ebstein, "D. Arch. f. klin. Med.," LVIII, p. 1.
[3] "Nothnagel's Handb.," vol. I, p. 220.

the twenty-fifth day, deep ulcers were found, some of them partly healed.

Mercury is said to have the same effect as lead on the sound-perceiving apparatus of the ear.

Copper, antimony, and phosphorus are said to produce hoarseness, but the manner of its production is nowhere indicated. Hemorrhages into the pharyngeal structures, especially the tonsils, have been observed in phosphorus-poisoning.

Copper and phosphorus, as they are used in the arts, may set up acute rhinitis and effect alterations in the septum analogous to those produced by chromic acid (Seifert).

In a case of poisoning by silver nitrate in a man who worked with the substance in a glass pearl factory, v. Jaksch found patches of bluish-black pigmentation on the external skin, on the mucous membrane of the mouth and tongue, on the drum membrane, and on the laryngeal mucous membrane.

The medicinal use of compounds belonging to the aromatic series is often followed by disturbances in the auditory sphere, even when the maximum dose is not exceeded. The upper air-passages usually escape. In a very few cases erythema (quinin, antipyrin, and salicylic acid), pemphigoid eruptions (antipyrin [2]), or hemorrhages were found on the mucous membrane of the pharynx as on the external skin. (We find no mention of the larynx in this connection.)

We may mention a few very unusual observations, such as a case, reported by Ebstein,[3] of intoxication in an employee of a salicylic acid factory, in which, in addition to marked pharyngitis, there was edematous swelling about the vocal processes and in the trachea; Hilbert [4] remarks that after the use of antipyrin and antifebrin he has observed the occurrence of parosmia consisting in the smelling of aromatic flavors, such as cinnamon; and, finally, the occurrence of an acute edematous angina after the use of salol—a statement for which Lavallée [5] is responsible.

In strychnin-poisoning there is marked hyperesthesia of the auditory nerve, which may, under the influence of an auditory impression, lead to general convulsions.

[1] Nothnagel's "Spec. Path. u. Therap.," vol. I, p. 240.
[2] Veil, "Arch. f. Derm. u. Syph.," 1891, p. 33.
[3] "Wien. klin. Wochen.," 1896, No. 11.
[4] See "Semon's Centralbl.," VIII, p. 558.
[5] See "Semon's Centralbl.," VIII, p. 380.

Quinin, salicylic acid, and antipyrin give rise to tinnitus aurium and difficult hearing. The toxic effect of the two first-named substances is generally recognized, and has been studied experimentally; we possess investigations by Weber-Liel and his followers as well as by Kirchner which establish beyond a doubt the occurrence of clinical and anatomic disturbances in the organ of hearing. According to these investigations, the effect of quinin and salicylic acid are very much the same, except that the disturbances after excessive use of salicylic acid are more violent and somewhat more persistent. The administration of 1 gm. of muriate of quinin and from 4.5 to 5 gm. of sodium salicylate was followed after from one to one and one-half, and from two and one-half to four hours respectively, by various subjective noises in the ear, which had completely disappeared twelve hours later; while after the use of salicylic acid the subjective symptoms lasted several days. The effect on the hearing occurred somewhat later than the tinnitus aurium, and always lasted longer, but after salicylic acid it persisted for several months.[1] We learn something of the way in which the auditory disturbance is produced by Kirchner's experiments on animals; after giving quinin (1.0) and sodium salicylate (2.0), he found ecchymoses in the mucous membrane of the tympanic cavity and of the vestibule, showing that the phenomenon is due to a disturbance of blood pressure, and not to a direct toxic effect on the organ of hearing. In view of the possibility of extravasations occurring in the labyrinth, where absorption is imperfect and the functional disturbances which result are therefore lasting, the greatest caution is indicated in prescribing this remedy for persons who have ever been subject to ear disease; Weber-Liel found that the impairment of hearing which follows the use of these remedies lasts much longer, and may even become permanent, in persons subject to ear disease.

Poisoning with sausage and fish, due to the presence of ptomains containing a number of alkaloid bodies, produces dryness of the mucous membrane and hoarseness similar to that observed in atropin-poisoning. According to v. Jaksch, there may be symptoms of bulbar paralysis and pharyngeal and laryngeal palsies, but unfortunately we

[1] Weber-Liel, "Mon. f. Ohr.," 1882, p. 7.

have no laryngoscopic findings, and Stuffer remarks on the difference of opinion as to the occurrence of hoarseness in fish-poisoning.

It has been said by Moos and Hackley that chloroform narcosis may be accompanied by deafness, tinnitus aurium, and double hearing, and Haug reports having seen such disturbances very frequently. But as the tinnitus aurium, paracousis, and auditory hallucinations can be attributed to the narcosis, Haug's statement—especially with regard to auditory hyperesthesia, which "may persist for several hours or even several days after the narcosis," and with regard to double hearing—can not be accepted unreservedly in the absence of positive case histories. It appears to be true that a progressive diminution in the power of hearing occasionally follows narcosis, particularly after the patient has been repeatedly subjected to an anesthetic, but the etiologic factor concerned is very difficult of interpretation. I have often seen patients, particularly women, gradually develop a slowly increasing deafness, which is usually attributed to chronic middle-ear catarrh, several decads after they have undergone chloroform narcosis; but we should accord to the imagination of our patients a fatal influence on our science if we allowed such statements to pass as current.

Finally, we may mention the intoxications that follow the abuse of tobacco and alcohol. Here we have to deal both with a local irritant effect on the mucous membranes of the upper air-passages and with the general toxic effect on the system. The catarrh of the smoker and the alcoholic has become proverbial. We have all had ample opportunity to convince ourselves of its occurrence, and there can be no doubt on the subject. The question whether the combustion products which are mixed with the smoke or a specific quality inherent in the vegetable poison—of which nicotin is usually considered the prototype—is responsible for this irritation of the mucous membrane may perhaps find its answer in the results of investigations conducted on chewers and snuff takers, in whom, except for the mechanical irritation, the effect on the mucous membrane bears no proportion as to intensity and extension of the process to that observed in smokers, in spite of the fact that the tobacco is much more thoroughly and completely absorbed. Analogous to the extensive laryngeal catarrh, we have

catarrh of the tubes, with its consequences to the middle ear in the form of catarrhal otitis media, which offers the best explanation for the chronic hardness of hearing so frequently observed in smokers, and which usually presents the character of a simple middle-ear catarrh. The possibility of a chronic neuritis of the auditory nerve analogous to tobacco amblyopia, which Moss assumes to be the cause of the difficult hearing and tinnitus aurium, can not be denied; but, so far, we have no proof of its occurrence.

The effect of alcohol on the organ of hearing is well known. It leads to tinnitus aurium and difficult hearing of a progressive character. The chief etiologic factor given is chronic middle-ear catarrh secondary to chronic pharyngitis; in addition to which the effect of the alcohol on the vascular system and its stimulating psychic effect no doubt play an important part in the production of tinnitus aurium and hallucinations.

Hoarseness has been mentioned as a symptom of acute alcoholic poisoning, but since alcoholic paralysis of the recurrent nerve has never been described, it must be regarded as the result of a disturbance of coordination due to the intoxication.

Alt [1] had occasion to observe alcoholic neuritis of the auditory nerve in a case of alcoholic multiple neuritis.

In conclusion, I wish to add a caution in regard to the use of irritating and astringent remedies in the treatment of the nose, as such substances, especially when used in the form of a douche or a powder, are very likely to produce disturbance of the sense of smell. In this respect the zinc salts, alum, tannin, and carbolic acid are particularly dangerous, and should be absolutely avoided in the treatment of the nose.

[1] "Mon. f. Ohr.," 1897, p. 171.

XII. NERVOUS DISEASES.

1. GENERAL REMARKS ON DISEASES OF THE LARYNX IN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

DISEASES OF THE SENSORY AND MOTOR NERVES OF THE LARYNX.

The disturbances which may occur in the larynx as a result of disease of the central nervous system are both sensory and motor. The pneumogastric nerve supplies the larynx with sensory fibers through the superior laryngeal nerve and its internal branch, which is distributed to the mucous membrane of the base of the tongue, the epiglottis, the pyriform sinuses, and the entire interior of the larynx; hence, disease of the sensory nuclei and roots of the pneumogastric may produce sensory disturbances in the form of anesthesia, paresthesia, and hyperesthesia. These are most frequent in bulbar disease and in diseases affecting the trunk of the vagus, and are therefore of great importance for the diagnosis of these conditions. They have also been occasionally observed in hemiplegia, in cerebral focal diseases, and in progressive paralysis, but have only a historic interest in this connection.

The motor disturbances in the larynx manifest themselves as irritative motor phenomena, as disturbances of coordination, and as palsies. The irritative phenomena occur in the form of tonic spasms, which are designated spasm of the glottis, laryngeal crises, and ictus laryngis; or in the form of clonic spasms, as rhythmic twitchings and tremors or atactic movements of the vocal cords, such as are sometimes observed in cases of brain tumor, cerebral abscess, and meningitis, and particularly in multiple sclerosis, in bulbar paralysis, and in a great number of neuroses.

Paralysis may occur either in the groups of muscles sup-

plied by the superior laryngeal or in those supplied by the inferior laryngeal or recurrent nerve. The superior laryngeal nerve, through its external motor branch, supplies the cricothyroideus muscles, whose function it is to make tense the vocal cords. Paralysis of this muscle, which manifests itself in roughness of the voice and sagging of the vocal cord on the paralyzed side during phonation, and by a slight waviness of the free border of the vocal cord, occurs very rarely as the result of an isolated paralysis of the external branch of the superior laryngeal nerve, and is never the result of a central lesion. When a paralysis of the cricothyroid muscles occurs in connection with a general paralysis of the motor fibers of the pneumogastric, either of central or peripheral origin, involving both the inferior and superior laryngeal nerves, it becomes merged in the general picture of complete paralysis of the vocal cords, and can not be distinguished clinically from that of paralysis of the recurrent nerve alone.

Paralysis of the recurrent nerve is by far the most important from a diagnostic point of view, for it is the typical symptom of a lesion of the motor paths in the central nervous system, whenever peripheral disease of the nerves or injury to the nerve-trunk can be excluded. As the question of paralysis of the recurrent nerve, its origin, and the interpretation of the laryngoscopic image which it produces has been and still is the subject of numerous controversies, it may not be out of place to present the *present state of the question of paralysis of the recurrent nerve.*

The muscles of the larynx that exert any influence on the movements of the vocal cords are divided into three groups, named, respectively, the openers, closers, and tensors of the rima glottidis. Opening of the glottis is effected by the crico-arytenoideus posticus drawing the muscular process of the arytenoid cartilage to which it is attached inward and at the same time rotating the vocal process outward. Closure of the glottis is accomplished by the combined action of various muscles which together make up a muscular ring embracing the entire glottis. Each one of these small muscles has its peculiar action, and the cooperation of all is required to effect exact approximation of the vocal cords. Finally, there is a third group of muscles, which connects the cricoid with the thyroid cartilage, and whose function it is to stretch the vocal

cord by increasing the distance between the vocal process and the anterior angle of the thyroid cartilage. As the resulting posterior displacement of the plate of the cricoid cartilage is accompanied by depression, the vocal cord, when stretched in this way, occupies a deeper position.

These three groups of muscles are supplied by the inferior, or recurrent, and the superior laryngeal nerve. Now, it is a remarkable fact that the openers and closers of the glottis, although mutually antagonistic, are both supplied by the inferior laryngeal nerve, while the motor branch of the superior nerve exclusively supplies the tensor muscles. It follows that any sudden injury to the inferior laryngeal nerve affects the openers and closers equally, so that the vocal cord, in the absence of antagonistic muscular traction, assumes a position of equilibrium—a position, in short, which is designated "the pathologic cadaveric position." The term "cadaveric position" was first used by v. Ziemssen, because it was found that the position of the vocal cords postmortem was the same as that seen in paralysis of the recurrent nerves. In both cases the vocal cords assume a position midway between inspiration and expiration. In recent times it has been repeatedly pointed out that the width of the glottis is not exactly the same in both cases, and that in the pathologic cadaveric position due to paralysis of the recurrent the vocal cords are slightly more adducted than in the so-called genuine cadaveric position, as seen in the dead body. This variation is to be attributed to the action of the crico-thyroid muscles, which are not affected by paralysis of the recurrent nerves, as they receive their innervation from the superior laryngeal, and can therefore continue to act in a peripheral palsy of the recurrent nerves. We know that the action of these muscles consists in stretching the vocal cords and at the same time in slightly approximating the edges of the vocal cords to the median line. This phenomenon is found to be retained in the pathologic cadaveric position, and explains the difference between the two kinds of cadaveric position. How the function of the cricothyroid muscle is affected in central palsies is not known, but it is probable that its motor nerve has the same origin as the other nerves, so that it must be held to be involved in any central paralysis of the vocal cords.

In addition to this complete paralysis of the recurrent

nerve, which affects the adductors and abductors equally, there is another important form of paralysis affecting this nerve, which is designated *posticus paralysis.* In the laryngoscopic image the vocal cord is seen to be immovably fixed in the median line, while the free border is taut, instead of concave, as in paralysis of the recurrent, so that phonation remains normal. This median position is explained by the failure of the abductors, and the condition is therefore designated posticus paralysis. It is this posticus paralysis that has given rise to so many controversies, which have again been revived in recent times, and are still very active. In order to understand the question thoroughly we must premise Semon's proposition, which says: "In organic progressive diseases of the roots and trunks of the spinal accessory, pneumogastric, and recurrent nerves the dilator fibers are affected earlier than the constrictor fibers, or may even be attacked exclusively." Applied to actual practice, this means that in such progressive diseases of the recurrent nerve we have first a paralysis of the posticus, and later paralysis of the adductors of the vocal cord, such as have just been described as total paralysis of the recurrent nerve. The correctness of this law, which is known as Semon's law, has been subjected to a rigorous test by Semon himself. He first formulated it on the basis of a series of clinical cases, and has since confirmed it in various publications by adducing physiologic and etiologic facts in its support. But in spite of these positive proofs the law has not been accepted, and many animated controversies have taken place between Semon and his followers on the one hand, and his opponents on the other. I am forced to go into this matter in some detail, as an exact understanding of the entire question is necessary in the criticism of the voluminous literature which has appeared on the subject. The question of this primary posticus paralysis of Semon's is important, because it enables us approximately to judge of the duration of a paralysis by observing whether the affected vocal cord is in the median or in the cadaveric position, and because it is a sign that the primary disease is progressing if the posticus paralysis, in spite of treatment, goes on to complete paralysis of the recurrent nerve.

I shall divide this discussion of the median position of the paralyzed vocal cord which has been designated as posticus paralysis into two sections; for, in the first place,

the question must be settled whether a median position of the vocal cord necessarily means that there is an isolated paralysis of the crico-arytenoideus posticus, and, in the second place, we must attempt to explain how, when the fibers that supply the antagonistic muscles are contained in the same nerve-trunk, those which innervate the crico-arytenoideus posticus can be for years the only ones affected by the paralysis.

According to Krause, who bases his opinion on experimental investigation, a median position of the vocal cord may, under certain conditions, not as yet very well explained, be due to reflex contraction of the laryngeal muscles. Krause experimented on animals by slowly constricting the recurrent nerve under proper precautions, and observed that a median position very soon appeared, which after about twenty-four hours changed to the cadaveric position. He accordingly adopts the theory, which he explains with much ingenuity, that the gradually increasing irritation of the nerve first gives rise to a reflex contraction, which first manifests itself in a median position of the vocal cord, because the adductors surpass all the other muscles in bulk, but which finally goes on to the cadaveric position when the nerve is completely paralyzed.

The promulgation of this theory, which is accepted by various authors, was followed by another, recently advanced by Grossman, to the effect that total paralysis of the recurrent is not a cadaveric position, as is generally supposed, but rather a position of adduction near the middle line, which practically (Grossman is not very clear on this point) corresponds to the median position. The final cadaveric position is, according to him, the expression of an additional paralysis of the cricothyroid muscle, the occurrence of which he explains as the result of secondary atrophy of the antagonistic adductor muscles, due to disuse in consequence of the paralysis.

Although at first sight both Krause's and Grossman's hypotheses may appear plausible, they will not bear the test of careful examination, and are in direct contradiction to a great number of clinical and experimental facts. To give all my reasons for this difference of opinion would lead me too far astray, but I will mention a few facts of pathologic anatomy which are insisted on by various authors: As against Krause's hypothesis we have many cases in which

the picture of a posticus paralysis was seen *in vivo*, and where, after death, only the crico-arytenoideus posticus presented an atrophy which was too pronounced to be reconciled with the theory of muscular contraction, in view of the long duration of the posticus paralysis and the integrity of the adductor muscles.

In refutation of Grossman's hypothesis we have, in addition to many other considerations, the anatomic fact that it has so far been impossible to demonstrate positively the occurrence of atrophy of the cricothyroids in a simple paralysis of the recurrent nerve, where the superior laryngeal was positively excluded—a condition which is absolutely necessary to demonstrate atrophy due to disuse, as claimed by Grossman.

I therefore assume that I have disposed of these objections, and that in the form known as posticus paralysis we have actually to deal with an isolated paralysis of the crico-arytenoideus posticus muscle. As, therefore, paralysis of the abductors is the first sign of a slowly progressing injury to the recurrent nerve, we are confronted with the most inexplicable phenomenon when we consider that this nerve innervates not only the paralyzed muscle, but also its antagonists, the adductors. Hence, the discussion is practically narrowed down to the question as to why the dilators become paralyzed before the closers, in spite of the fact that both are supplied by the same nerve.

The explanation that the fibers destined for the posticus muscle are more superficial than those which supply the adductors can not be taken seriously, but I may mention the attempted explanation, which is based on Exner's experiments on animals. Exner and his disciples have devoted much study to the innervation of the larynx, and have found certain individual variations; most muscles appear to have a double innervation, either the corresponding nerves on both sides or several nerves of the same side being concerned in the innervation of one muscle. If this condition occurred regularly in man, we should naturally be led to conclude that in cases of isolated paralysis of the posticus, although there is a total paralysis of the recurrent fibers, the paralysis affects only the dilators, because in such a case the adductors derive an additional supply from another nerve. Unfortunately, this hypothesis is contradicted by a great number of clinical observations, as we have absolutely

no proof of individual variation in the form of a double innervation; besides, many of Exner's experiments are wanting in clearness, and other experimenters have not been able to confirm his results.

I recently, for a different purpose, practised extirpation of the various laryngeal nerves in rabbits, and in every case I observed atrophy of the abductors and adductors after division of the recurrent nerve, so that I am forced to exclude the existence of a double innervation for these cases. On the other hand, in a series of experiments which go to prove that there is a physiologic difference in the biologic relations of the two groups of muscles, we have exact and incontestible proofs of the greater vulnerability of the dilators, which might be responsible for a primary paralysis of the postici. It is proved by one series of experiments that the electric irritability of the posticus muscle disappears long before that of the adductors, and this condition is found not only postmortem, but also in ether anesthesia and when the nerve is gradually allowed to freeze. In this connection it is important to remember Grabower's discovery that the nerve-endings in the abductors differ morphologically from those in the adductors. From this we may conclude that the adductors and abductors are not ordinary antagonists, like the extensors and flexors of the extremities, and we must try to find some cause for their physiologic difference. This difference is found in their function, since the adductors of the vocal cords are concerned in phonation, while the abductors merely represent respiratory muscles. Corresponding to these different functions there must be different kinds of fibers in the recurrent nerve, some of which are intended for phonation while others transmit reflex impulses connected with respiration.

The question whether the action of the crico-arytenoideus posticus is exclusively a reflex action has been carefully studied by Semon and Horsley. These authors found that the ordinary respiratory position in which the glottis gapes wider than in the cadaveric position must be regarded as a reflex tonic spasm, which is constantly present in the posticus muscle under the influence of the respiratory center, its object being to keep the glottis sufficiently dilated for the act of respiration. The existence of such a reflex tonic spasm in the nerve-fibers destined for the posticus would serve to explain the physiologic fact previously mentioned,

that the abductors of the vocal cords become fatigued earlier than the adductors, since on this supposition we have to deal with two kinds of nerves in the recurrent—afferent and efferent nerves. The afferent nerves produce reflex tonic spasm in the posticus muscle, while the efferent nerves act as simple motor nerves to the adductors. Thus, the early and isolated appearance of paralysis of the posticus is best explained by the physiologic law that the irritability of afferent nerves is exhausted earlier than that of efferent nerves.

To sum up, we have learned that there are two kinds of paralysis of the recurrent nerve ; the first stage of a lesion to the recurrent nerve gives rise to posticus paralysis, while a fully developed recurrent paralysis finds expression in the so-called pathologic cadaveric position. We have left to consider only the mode of transition from one form to the other, and we learn from clinical observation that this takes place in a typical manner. The first step in the progress of the paralysis consists in a relaxation of the free border of the vocal cord, which was tightly stretched in the simple posticus paralysis. The border becomes concave toward the median line, then gradually bows outward, and finally goes on to recurrent paralysis. If recovery takes place in a recurrent paralysis, as I saw lately in a capital case of postdiphtheric paralysis, the vocal cord first moves into the median position, and for a short time presents the picture of a posticus paralysis, before it regains its normal movability.

THE LOCALIZATION OF CENTERS FOR THE MOVEMENT OF THE VOCAL CORDS IN THE CENTRAL NERVOUS SYSTEM, AND THE EFFECT OF DISEASES OF THE CENTRAL NERVOUS SYSTEM.

Hemorrhages, foci of softening, pseudobulbar paralysis, sclerotic foci, tumors, tubercular and syphilitic tumors, and cerebral abscesses may produce paralysis of the vocal cords when the lesion is situated in the central motor paths for voice production and for the movements of the vocal cords. The localization of movements of the vocal cords in the cerebrum is still a matter of dispute, so that the diagnostic value of paralysis of the vocal cords for the localization of such diseases is limited.

The number of cases of which we possess a clinical and

anatomic description is too small to afford a basis for a definite symptomatology of laryngeal disturbances in diseases of the central nervous system, and if I were to take up the various brain diseases individually, my description would be nothing more than an incoherent series of facts repeated from the literature. I shall therefore content myself with a short presentation of the views which prevail at the present time in regard to the localization of laryngeal movements in the central nervous system. This will form a basis in any given case for deducting the site of the morbid focus from the existing disturbances in the larynx.

The motor paths for the larynx in the medulla oblongata are better known than those in the cerebrum. The nuclei become typically involved in certain systemic diseases, and sensory as well as motor disturbances of the larynx result. But even in this region, although the question in the main is fairly well settled, there are certain points which are still under dispute, the most important one of which is whether the nucleus of the motor nerves of the larynx is to be found in the vagus or in the spinal accessory. After we have given a general description of the localization of the larynx in the cerebrum and its relation to the medulla oblongata, it will be necessary to discuss a few diseases of the spinal cord which give rise to typical disturbances in the larynx as a part of their general symptom-complex.

The motor nerves of the larynx are the superior and inferior (or recurrent) laryngeal nerves—branches of the vagus. The trunk of the vagus, therefore, contains the peripheral paths which transmit nerve impulses to the vocal cords and cause them to open or to close the rima glottidis in the service of the phonatory and respiratory function of the larynx. As the larynx has a double function—that of phonation, which is purely motor and is dependent on the will, and that of respiration, which consists in the reflex opening of the glottis under the influence of the respiratory center—there must be two different centers for adduction and abduction in the central nervous system. For the voluntary movements performed during speech we must assume, in addition to the center in the medulla oblongata, a second center in the cortex, while the reflex opening of the glottis during respiration, which takes place independently of the will, is probably but little, if at all, under the influence of the cortical center.

We emphasize this point because an impression has lately gone abroad that the vocal cords can be voluntarily adducted or abducted on one side. This view accords with the conception of a bilateral symmetric movement only so far as respiration is not altogether reflex, but partly subject to the will, since we are able to make deep voluntary inspirations and thereby effect a wide gaping of the rima glottidis. We learn from experimental investigations and from pathology that the assumption of two separate centers for the two kinds of movement is necessary to explain the occurrence of the different kinds of paralysis.

It must be admitted at the outset, however, that the discussion is only in its infancy, and that, owing to the contradictory statements and findings of careful observers and experimenters, it is impossible to give a clear objective presentation of the state of affairs, so that in attempting to explain the various phenomena which present themselves we are often forced to resort to hypotheses to bridge the gaps in our argument.

Even the question of the origin of the motor nerves of the larynx is not definitely settled. The controversy as to whether the nucleus of the vagus or that of the spinal accessory, or both together, represents their origin has been going on for several decads, and has been lately rekindled by Grabower's investigation, just as the authorities were beginning to incline to the opinion that the motor fibers for the larynx were derived from the nucleus of the spinal accessory.

Grabower has proved by a series of sections through the medulla oblongata and the spinal cord that the spinal accessory is a purely spinal nerve; its nucleus and deep roots have no relation to the nucleus of the vagus. According to him, the ventral nucleus of the vagus (the nucleus ambiguus) represents the origin of the motor nerves of the larynx. In a detailed discussion of this question Semon [1] says that his clinical experiences have been such that he can not agree with Grabower's opinions, because he is unable to reconcile them with certain observed cases of simultaneous paralysis of the vocal cords and of the trapezius and sternocleidomastoid muscles, which are supplied by the spinal accessory nerve. But Grabower [2] himself denies

[1] Heymann's "Handb. d. Laryng.," vol. I, p. 606.
[2] "Arch. f. Laryng.," vol. V.

that those cases in which there is a simultaneous paralysis of the larynx and of the spinal accessory are any proof that these various groups of muscles are under the control of a common nucleus. Hence, we have to regard the question as still undecided for the present. We may mention that Claude Bernard has advanced the opinion that the spinal accessory represents the nerve of phonation, and the vagus the nerve of respiration.

While the occurrence of bulbar paralysis in the larynx, in diseases which affect the bulbar nuclei, is definitely established, the question whether cortical paralysis of the larynx ever occurs is still undecided. It is known that Krause found a spot on the anterior lower extremity of the anterior central convolution, immediately behind the precentral fissure,—called after him, "Krause's center,"—irritation of which on one side of the brain produces a bilateral adduction of the vocal cords. It follows, therefore, that each of these two cortical centers for adduction is capable of influencing the movements of both vocal cords; but, on the other hand, unilateral lesion of this cortical region is not capable of suspending movement in the larynx, as has been shown by extirpation of these parts. In view of the great frequency of apoplexy and of other lesions in this region of the cortex, the literature ought to contain a great number of cortical palsies, but, as a matter of fact, we possess only a very small number of observations,[1] which can not even be definitely referred to a unilateral cerebral injury, because no autopsies are given and the clinical history is not quite clear. On the other hand, when the centers on both sides of the cerebrum are diseased, cortical palsy undoubtedly results, as was proved by Semon,[2] both by experiments on animals and by two cases where the diagnosis was confirmed by an autopsy.

In syphilis, tuberculosis, multiple sclerosis, and meningitis, and in tumors and hemorrhages, we should therefore expect a cortical palsy, affecting both vocal cords, due to injury of both Krause's centers.

A great number of theories, more or less well supported by experiments on animals, have been presented on this subject, while clinical observations, on the other hand, are

[1] Cases bearing on this question will be found quoted by Onodi, "Rev. hebd. de laryng.," etc., 1898, No. 4.

[2] Heymann's "Handb. der Laryng.," I, pp. 692 and 701.

very meager. If all these observations were perfectly clear there would be no controversy, but as some authors claim to have seen a crossed unilateral cortical paralysis of the vocal cords, while others absolutely deny that any but a bilateral cortical lesion is capable of producing a double paralysis of the vocal cords, there is evidently room for a great deal of discussion, and any contributions, such as those which have lately been added by Uchermann,[1] are well worthy of attention. In connection with a case of right-sided hemiplegia, motor aphasia, and paralysis of the adductors in the larynx, he raises the question whether a one-sided—that is to say, left-sided—injury of the phonation center is capable of producing a double palsy of the adductors, and suggests the possibility that the center of phonation, like that of speech, is usually located on one side. Injury of the fibers which pass through the internal capsule from the cortex to the medulla oblongata necessarily produces the same effect as a cortical lesion.

The existence of a center of phonation in the posterior corpora quadrigemina, and the corresponding area in the floor of the fourth ventricle, capable of producing approximation of the vocal cords, even after communication with the cortex has been interrupted, is maintained by Onodi, and denied by Klemperer and Grabower.

According to Semon and Horsley, abduction of the vocal cords or opening of the rima glottidis is under the control of two different regions in the medulla oblongata: One of these is situated in the ala cinerea; the other, in the region of the origin of the auditory nerve, extending to the mouth of the aqueduct of Sylvius. Irritation of these regions was always followed by bilateral abduction of the vocal cords.

[1] "Arch. f. Laryng. u. Rhinol.," p. 332.

2. GENERAL REMARKS ON THE AURAL DISTURBANCES PRODUCED IN DISEASES OF THE CENTRAL NERVOUS SYSTEM.

THE MECHANISM OF FUNCTIONAL DISTURBANCES IN THE EAR AND THE ELECTRIC REACTIONS OF THE AUDITORY NERVE.

The functional disturbances produced in the organ of hearing by disease in the central nervous system consist in disturbances of the hearing or in the equilibrium, according as the paths of the cochlear or those of the vestibular nerves are involved. When the trunk of the auditory nerve is diseased, both hearing and equilibrium are affected. Nervous disturbances of the hearing in central disease manifest themselves either in abnormal excitability of the auditory nerve, which may be so intense that the perception of certain tones becomes positively painful, or in torpor of the nerve, which, again, may go on to complete insensibility to auditory impressions. This form of deafness is accompanied by certain characteristic pathologic alterations in the ear which enable us to distinguish it from those disturbances having their seat in the sound-conducting apparatus. The hearing in such cases is lost for certain tones, so that in testing the field of hearing one is forced to use a long series of graduated tuning-forks. The tuning-fork test is intended to determine the power of the auditory nerves to perceive tones which reach the internal ear through the air or through the craniotympanic conducting path. If craniotympanic conduction is very much weakened or entirely absent, it is a sign of nervous disease.

Tinnitus aurium is the first symptom observed in disease of the auditory nerve paths. In the first stage of a disease in the central nervous system it may be the expression of irritability of the auditory nerve, but it may also occur in the later periods, in which the irritability of the nervous paths is entirely lost. These subjective noises present certain characteristic qualities, which serve to distinguish them from those produced in the middle ear; they may be continuous and low in pitch, and so intense as to be compared with the thunder of cannon or the din of a railroad train; they may be high-pitched and musical; the tinnitus aurium may have a register or pitch of C^4 to C^5; or it may be a

musical or harmonious sound described as a melody, the ringing of bells, or the twittering of birds.

Finally, we have the disturbances of equilibrium which are regarded as the expression of disease of the vestibular nerve, and which are often associated with nausea and vomiting.

These three symptoms are described together under the name of Ménière's symptom-complex. Before the purely symptomatic nature of these phenomena—which are common to all diseases of the auditory nerve paths—was appreciated, it was customary to speak of a Ménière's disease, because Ménière had first observed these symptoms in a case of sudden hemorrhage from the labyrinth. Since the appearance of v. Fränkel Hockwart's [1] publication, in which he presents this Ménière's symptom-complex in its true light for the benefit of the nonspecialist, it is to be hoped that the term Ménière's disease, as applied to the most various diseases of the nervous hearing apparatus, will be discarded altogether.

The functional disturbances just described do not enable us to determine the exact location of the disease in the auditory paths. They are simply diagnostic of a nervous disturbance of the hearing, and, so far as our present knowledge of these disturbances goes, we are unable to determine whether the peripheral terminations of the auditory nerve in the labyrinth, the nerve-trunk, or the central nervous paths are diseased.

To discuss these symptoms in detail would lead us too far into the domain of physiologic research concerning the special functions of the individual portions of the ear; we must, however, devote some attention to the electric examination of the auditory nerve, which has reached a high degree of perfection in the hands of nerve specialists, and is now universally used by them as a method of examination, while ear specialists even now stand skeptically aloof, just as they did thirty years ago, and continue to doubt the importance of a method which is insisted on by a great number of writers on neurology.

As the results of electric examination of the auditory nerve have been applied in various ways to diseases of the central auditory paths,—although it is, as a rule, very

[1] Nothnagel's "Spec. Path. u. Therap.," XI.

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imperfectly described or entirely omitted in text-books on otology,—I feel impelled to present a general résumé of *the significance of electric reaction of the auditory nerve.*

The attempt has been made to utilize the electric irritability of the auditory nerve for purposes of diagnosis and therapeutics; but the results in either direction have not been such as to justify the expectations raised by the dogmatic teachings of Brenner, published thirty-five years ago in his "Elektro-otiatrik." Brenner gives a normal formula for the healthy individual as follows:

Ka. Cl.	R″.	Very loud ringing.
Ka. D.*	R ∞.	Ringing persists during continuance of current.
Ka. O.	Nothing.	
An. Cl.	Nothing.	
An. D.	Nothing.	
An. O.	R′ >.	Louder ringing, gradually dying away.

(*During passage of current.)

His most important results are embodied in the propositions[1] that: "The cathodal contraction produces auditory sensation when the circuit is closed, and also during continuance of the current, but not when the circuit is opened. The anodal contraction gives no reaction either when the circuit is closed or during the continuance of the current, but does give a reaction when the circuit is opened. The anodal reaction *ceteris paribus* is weaker than the cathodal reaction. The cathodal reaction occurs immediately, the anodal reaction only after the current has lasted a certain time. After a short duration of the current, opening is not followed by a reaction at the cathode. The cathodal reaction becomes markedly increased immediately after closure, a phenomenon described by the patients as an echo, for they frequently remark that the echo is stronger than the first (or closing) sound. The reaction persists for some time, with a lessened intensity,—'reverberating echo,'—and then completely disappears, although the strength of the current remains constant."

The publication of these statements was, of course, received with equal interest by ear specialists and by neurologists, and gave rise to numerous control investigations. Schwartze[2] deserves the credit of being the first to find

[1] "Virch. Arch.," 28, p. 207.
[2] "Arch. f. Ohr.," 1, p. 44.

flaws in Brenner's formulæ, which materially diminished the value of the electric reactions for the diagnosis and treatment of aural diseases. His objections amount to the following proposition: That Brenner's normal formula for the reaction of a healthy auditory nerve is not by any means constant in persons with normal hearing; that Brenner's normal formula is given in absolute deafness, which can be only due to a disease of the nervous apparatus; and, finally, that, as far as treatment is concerned, the restoration of the normal "formula of reaction" has no effect on the power of hearing. These objections were answered by Brenner [1] and Erb. [2] At first Erb said [3] that "those who deny the existence or correctness of Brenner's discoveries are simply mistaken," but later he modified his opinion to the extent of confirming the first of Schwartze's objections. Although opinions in regard to the diagnostic value and the production of the reaction are now fairly well settled, the skeptical attitude adopted by Schwartze has in the main been justified, and the value of "Elektro-otiatrik" is not nearly so great to the ear specialist as would appear from the statements of neurologists.

The electric examination is conducted in two different ways, called the internal and external methods. The former was employed by Brenner, who filled the external auditory meatus with water, and then introduced an electrode, with certain precautions, [4] so that its extremity was rigidly held at a certain distance from the ear-drum and from the walls of the meatus. The other electrode was applied to the mastoid process, the forehead, the nape of the neck, the trunk, or the extremities. The external method introduced by Erb is the one now exclusively employed. It consists in applying an ordinary flat electrode—the cathode—in front of the tragus (taking care not to press on the tragus and thereby close the auditory meatus, as this would give rise to buzzing and humming noises), while the other electrode—the anode—is placed on the nape of the neck or the palm of the hand. A third method, in which the electrode is applied to the auditory meatus, filled with water (Brenner, Erb), presents no special advantage, while the

[1] "Virch. Arch.," XXXI, p. 483.
[2] "Arch. f. Augen- u. Ohrenheilk.," vol. I, p. 156.
[3] "Arch. f. Augen- u. Ohrenheilk.," vol. I, p. 158.
[4] "Virch. Arch.," XXXI, p. 493.

results of the examination are no more satisfactory when one of the electrodes is replaced by a silver wire introduced through a tubular catheter into the middle ear, as proposed by Wreden.[1]

The question has been raised whether the auditory nerve or its terminations are really excited by the electric current, or whether what is designated as the reaction of the nerve may not be due to the irritation of other structures in the ear. It was alleged that the reaction may be produced by contraction of the internal muscles of the middle ear (Schwartze, Wreden), by irritation of the sympathetic (Benedikt), or by a reflex irritation of the auditory through the trifacial nerve. While it has long been known that the bony labyrinth is a bad electric conductor, the question was again discussed by Gärtner and Pollak,[2] who declared, after a series of investigations on pathologic organs, that the electric irritability of the auditory nerve depends on the excitability of the nerve itself to an electric current and on the resistance met with in the ear.

I have not the space to discuss in detail the various arguments which have led to the adoption of the view that the condition of the auditory nerve itself determines the results of the reaction, without entirely disregarding the modification in the resistance due to hyperemic and secretory processes ; but in order to elucidate the present status of the question, I shall cite the propositions promulgated by Gradenigo,[3] which most nearly correspond with the results of practical experience in the healthy and in the diseased ear.

1. The normal ear gives an electric reaction of the auditory nerve only in exceptional cases and when the electric current is unusually strong.

2. There is a heightened irritability in all inflammatory and hyperemic diseases of the external, middle, and internal ear, and in the initial stages of a central cerebral disease.

3. The mode of reaction of the auditory nerve to the electric current is analogous to that observed in the other sensory and motor nerves.

In regard to the first proposition, it may be remarked that even the earliest followers of Brenner's doctrines became more and more reluctant to designate the acoustic

[1] "Petersb. med. Zeitschr.," 1891 ; reported in "Arch. f. Ohr.," VI, p. 147.
[2] "Wien. klin. Wochen.," 1888, Nos. 31, 32.
[3] "Arch. f. Ohr.," XXVII and XXVIII.

reaction as the normal formula to be aimed at in the healthy individual. Schwartze was not able to obtain the reaction in every case; later, Gradenigo found it present in only from 5% to 12% of normal ears, and then only when a higher current strength was employed, usually from 10 to 16 m.a., certainly never under 6 m.a. This agrees with Erb's [1] more recent publication, in which he says that galvanic stimulation of the auditory nerve is not always possible. As a very strong current is requisite to obtain the reaction in healthy individuals, the examination is usually attended with very unpleasant concomitant phenomena, such as vertigo and flashes of light.

It is therefore better to assume that there is a pathologic condition of the nerve whenever there is hyperesthesia to the galvanic current and not to attempt to lay down any normal reaction for healthy individuals. Daily experience shows that electric excitability is not common in persons the subjects of ear disease. Gradenigo found that he could usually obtain the reaction in 66% of cases of ear disease with a current strength of from 1 to 3 m.a., and always with a strength of less than 6 m.a. Although this increased excitability of the auditory nerve to weak currents undoubtedly points to pathologic processes in the organ of hearing, it has no special diagnostic significance, as it may occur in a great variety of diseases both of the ear and of the nervous system. It is much to be desired that ear specialists might contribute more to the investigation of electric excitability of the auditory nerve, in the hope of obtaining some definite diagnostic points; for the prevailing theory that the auditory nerve reacts readily in those diseases of the middle or the internal ear that are accompanied by intense inflammatory processes, but fails to react after the inflammation has subsided, and that the reaction of the nerve in acute or chronic exudative or nonexudative catarrh of the middle ear and in cases of gradual extension of such disease to the internal ear does not differ from that which occurs under normal conditions (Gradenigo),[2] is in urgent need of further elucidation, as it is in direct contradiction to other observations, especially those published by Erb,[3] which are in every way admirable.

[1] Ziemssen's "Handb. der allgem. Ther.," III, 1882, p. 236.
[2] "Arch. f. Ohr.," XXVIII, p. 247.
[3] Ziemssen's "Handb. der allgem. Ther.," III, 1882.

The power of hearing does not appear to bear any relation to the electric behavior of the auditory nerve. According to Gradenigo, the greatest value of galvanic hyperesthesia of the auditory nerve in diagnosis of central abscess of the nervous system is found in connection with brain-tumor; he found the phenomenon present in all but one out of 18 cases. In tabes dorsalis, multiple sclerosis, and chronic myelitis it is absent, according to Gradenigo, but is said to have been observed by Erb. Gradenigo points out that in subnormal sensitiveness to auditory impressions in hysteria the electric reaction of the auditory nerve is never increased.[1]

It is worthy of remark that ocular disturbances due to central or intracranial paralytic lesions in the domain of the organ of sight, such as ocular palsies and disturbances of the accommodation, are associated with galvanic hyperesthesia of the auditory nerve (Brenner, Erb). In ordinary disturbances associated with facial paralysis the electric conditions vary. In some cases there is hyperesthesia with paradoxic reaction (Remak);[2] a hyperesthesia was observed occasionally in cases of aural hallucinations (Jolly).[3]

Finally, it may be mentioned that certain alterations occur in the reactions of the auditory nerve which have been described as a paradoxic reaction and as a galvanic hyperesthesia, with anomaly and inversion of the normal formula. Paradoxic reaction consists in the production of sensations, corresponding to the indifferent electrode, in the ear which is not included in the circuit; this is regarded by Erb as the expression of so intense a heightening of the galvanic irritability of the auditory nerve that even the weaker loops of the current, which reach the ear not included in the circuit, are capable of producing the auditory sensation. In a case of complete left-sided deafness, with the remains of an old suppuration, Erb found the normal formula inverted, as follows:

Ka. Cl. . . . . —
Ka. D. . . . . —
Ka. O. . . . . p>' (piping sound, gradually disappearing).
An. Cl. . . . . R'.
An. D. . . . . P ∞ .
An. O.

[1] "Haug's Vort.," p. 411.
[2] "Grundriss der Elektrodiagnostik u. Elektrotherapie," 1895.
[3] "Arch. f. Psych.," 1894, IV.

I have seen in sclerosis of the middle ear with involvement of the internal ear cases in which the ear under examination presented the normal formula, while the ear not included in the circuit presented the paradoxic formula :

Right.		*Left* (included in circuit).
——	Ka. Cl.	R (ringing).
——	Ka. D.	R ∞ .
R.	Ka. O.	——
R.	An. Cl.	——
R ∞	An. D.	——
——	An. O.	R.

As an instance of other anomalies, Erb gives the following reactions, which occurred in a man fifty-four years old with chronic impairment of the hearing, tinnitus aurium, and opacity and contraction of the ear-drum.

Ka. Cl.	P′.
Ka. D.	P ∞ .
Ka. O.	b (buzzing noise).
An. Cl.	B′.
An. D.	B >.
An. O.	p >.

It is impossible to determine whether torpor of the auditory nerve is present or not, as the reaction in the healthy individual is not constant.

THE LOCALIZATION OF THE EAR IN THE CENTRAL NERVOUS ORGANS.

The origin and root-fibers of the cochlear and vestibular nerves, which together make up the auditory nerve, are twofold. While our knowledge of the former is fairly complete, thanks to the investigations of Held, Flechsig, and Bechterew, any description of the latter must be largely hypothetic. The fibers of the cochlear nerve, the peripheral endings of which are found in the cochlea, spring from the ventral auditory nucleus, and to a slight extent from the tuberculum acusticum.[1] A second system of fibers originates in the ventral auditory nucleus (accessory nucleus), and, after passing through the corpus trapezoides, extends to the superior olive of the same and of the opposite side. The lateral root represents the continuation of the cochlear tract to the posterior corpora quadrigemina. It is joined, however, by the fibers from the auditory

[1] After Edinger's description, p. 359, Fifth Edit.

tubercle, which run directly through the striæ acustica to the lateral root.[1] The lateral loop ends in the posterior quadrigemina. Each of the posterior corpora quadrigemina sends out fibers through the inferior brachia, both of the same and of the opposite side, to the internal geniculate body, where some of these fibers end. The remainder pass under the pulvinar into the internal capsule, where they divide into two bundles and are distributed to the transverse convolutions of the temporal lobe (superior temporal convolution). "One of these bundles ascends in the neighborhood of the external capsule and reaches the auditory sphere, while the other accompanies the optic radiation for some distance and, after passing around the inferior posterior portion of the fossa Sylvii, ascends to the transverse convolutions in the temporal lobe close to the second and third convolutions."[2]

The course of the vestibular nerve is very obscure. It appears to originate in the dorsal auditory nucleus or Deiter's nucleus, which lies to the mesial side of the restiform body. Its connections with the vermiform process of the cerebellum are not known.

From this description it follows that auditory disturbances may be expected in disease of the auditory nucleus in the medulla oblongata, of the superior olivary nucleus in the pons, of the posterior quadrigemina, and, finally, of the first (superior) temporal convolution, and in disturbances of the nervous paths which connect these nuclei. Tumors and abscesses, foci of softening in the brain-substance, tubercular and syphilitic disease, cerebral hemorrhage, and many other diseases of the central nervous system may produce a focal lesion by destroying the central pathways. The only symptom of such a lesion in the cochlear tract is difficult hearing, while vertigo and the signs of Ménière's symptom-complex generally are absent. Impaired hearing from a central cause is recognized by the presence of other phenomena of cerebral disease, and its gradual increase in a subject whose hearing had always been perfectly good corresponds to the gradual growth of the tumor. Unfortunately, we have no accurate knowledge concerning the nature of a central deafness localized in the cortex, but when the cause is found in a lesion of

[1] See illustration No. 247, Edinger.
[2] Flechsig, "Gehirn u. Seele," 1896, p. 75.

the tegmentum or mesencephalon, the resulting auditory disturbance presents certain characteristics, which have been described by Siebenmann. Bone conduction is very much impaired or entirely abolished. Weber's experiment is not regularly successful, and may be lateralized either to the healthy or to the affected side. In the beginning of the developing deafness perception is lost for the lower notes only, while later in the course of the disease all the notes of the scale become uniformly inaudible, so that finally the patient retains only the power of hearing a certain number of notes in the lower middle register, as in diseases of the labyrinth and of the auditory nerve.

Subjective ear noises are rarely observed. Hyperesthesia of the auditory nerve appears to be possible in the early stages of a lesion of the auditory centers; at least, this would seem to explain the increased electric irritability of the auditory nerve described by Gradenigo.

Oppenheim [1] quotes the statement that in tumor of the superior temporal convolution the epileptic attacks were preceded by an auditory aura. The important question as to which side is affected in unilateral lesion of the cerebral roots of the cochlear nerve has not yet been decided. The pathways cross each other at various points in their course through the pons, in the tegmentum, and in the corpora quadrigemina, but the decussation appears to be only partial, so that the cortical centers for hearing on both sides of the brain appear to be connected with both auditory nerves. The result of this arrangement is that unilateral disease in the region of the temporal lobe, where the cortical center for hearing is found, does not produce unilateral deafness of the opposite side (crossed deafness), as some authors have claimed. Permanent central deafness can be produced only by the destruction of the cortical centers for hearing in both hemispheres.

The significance of the posterior corpora quadrigemina in auditory disturbances has lately been carefully investigated by Weinland and Siebenmann. Weinland maintains that disease of one of the posterior corpora quadrigemina produces auditory disturbance on the opposite side; while Siebenmann, on the contrary, claims that a lesion of only one of the posterior corpora quadrigemina does not produce

[1] "Lehrb. der Nervenkrankh.," p. 94.

deafness—a statement which is in direct opposition to the generally accepted opinion that the corpora quadrigemina contain one of the auditory centers. By a careful review of the literature Siebenmann [1] shows that in all cases of deafness due to injury of the mesencephalon there is either compression or destruction of the tegmentum (or of the internal capsule), whereas in simple cases of tumor of the corpora quadrigemina the hearing remains intact. From this he argues that the auditory disturbance is not directly due to the situation of the tumor in the posterior corpora quadrigemina, but rather to its interference with the surrounding parts and to the compression of the adjacent portions of the mesencephalon, which contain the auditory pathways.

As we have just remarked, Weinland says that the loss of hearing occurs on the side opposite to that of the diseased corpora quadrigemina; Oppenheim believes that either the ear on the same side as the tumor or that on the opposite side, or even both ears, may be affected; while, according to Siebenmann, any lesion of the tegmentum produces bilateral deafness.

The auditory disturbances that have been observed in diseases of the cerebellum must be attributed to extension of the diseased focus to the medulla oblongata and pons, or directly to the trunk of the auditory nerve. Such a disease necessarily interferes with the roots and centers of the vestibular nerve contained in the cerebellum, but as we have no definite knowledge of the relation existing between this cerebellar ataxia and the static functions of the organ of hearing, the question will not be included in the present discussion.

It is often very difficult to distinguish an auditory disturbance due to central lesion from intracranial lesion of the trunk of the auditory nerve. A great number of cases are known in which the auditory nerve was included in tumors originating at the base of the brain, in the cerebellum, or in the pineal body. Such tumors even penetrate through the porus acusticus internus into the labyrinth. A differential diagnosis in such cases is impossible.

In the etiology of the auditory disturbances which we have just described we have so far considered only those

[1] "Zeitschr. f. Ohr.," vol. XXIX.

diseases which produce a direct lesion of the auditory pathway and its cerebral centers. In other words, we regarded the auditory disturbance as a direct result of such a lesion. We must now mention another pathologic condition, which is recognized by various authors, and to which Gradenigo, in Schwartze's "Handbuch der Ohrenheilkunde," [1] assigns a very important place, although its occurrence is now generally discredited: namely, the question of the *influence on hearing of a rise in the intracranial pressure.*

Reasoning by analogy from papillary congestion, it was natural to assume that increased intracranial pressure might exert some influence on the auditory nerve, as the conditions are in certain respects similar. Moos considered it doubtful that auditory disturbances could be due to increased pressure from cerebral tumors; Steinbrügge interpreted a depression of Reissner's membrane as dependent on increased intracranial pressure (an explanation which caused some discussion in the Naturf. Vers. in Heidelberg, the sense of the meeting being that the depression was simply an artifact); and Gradenigo assumes that "in cases of brain-tumor with increased intracranial pressure, a lymphatic infiltration occurs at the peripheral ending of the auditory nerve analogous to the papillary congestion of the optic nerve." This interpretation is very artificial and anything but unassailable, for most pathologists deny that papillary congestion of the eye is due to intracranial pressure alone, attributing it rather to toxic influences. Although histologists possess perfect methods and abundant material for the anatomic investigation of the eye, their results are not by any means uniform; how, then, can we expect to draw any reliable conclusion from the superficial descriptions of only two histologic examinations of the labyrinth, in the examination of which it has so far been impossible to exclude with certainty the fallacies of artifacts? It is therefore not to the credit of otology, and does not in the least add to our understanding of the question, to erect a hypothetic "papillary congestion of the auditory nerve" merely for the purpose of substantiating a preconceived opinion. In addition we may mention the conclusion reached by Asher [2] in a very careful work on the subject—that rise in the intracranial pressure does not produce any constant dis-

[1] Vol. II, p. 530.
[2] "D. Zeitschr. f. klin. Med.," 27, p. 513.

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turbances in the organ of hearing, as the pressure conditions in the endolymphatic and perilymphatic spaces, which depend on the hydrostatic pressure of the lymphatic fluid, tend to regulate each other mutually, and thus to prevent the occurrence of excessive pressure.

3. NERVOUS DISEASES WHICH PRODUCE DEFINITE ALTERATIONS IN THE NOSE, PHARYNX, AND LARYNX, AND IN THE EARS.

DISEASES OF THE SPINAL CORD.

Tabes Dorsalis.

The occurrence of laryngeal disturbances in tabes was formerly regarded as very rare, and until very recently opinions diverged as to the existence of any relation between tabes and difficult hearing. But now we have a long series of statistics and reported cases which prove that the vagus and auditory nerve are comparatively often involved in tabes dorsalis, if not quite as frequently as the optic nerve. According to Klippel,[1] the olfactory nerve also becomes involved in tabes, and there result disturbances of the sense of smell, manifesting themselves in unilateral anosmia, parosmia, and hallucinations of scent.

Statistics differ very widely as to the frequency of laryngeal symptoms in tabes dorsalis; Krause found motor disturbances in 13 out of 38 cases, but does not give any detailed description of their nature; Marina, on the strength of Fano's investigation, gives 19 cases of motor disturbances in 36 patients suffering from tabes, in all of which the conditions were found to be abnormal. Dreyfus found two cases of double posticus paralysis among 22 tabes patients; Burger [2] in 6 out of 20 cases found that motor disturbance could be demonstrated with the laryngoscope. I may add that among 27 tabetic patients in the Medicinische Universitäts-Poliklinik in Leipzig I found no disturbances in the larynx; while, on the other hand, in the case of one tabetic patient who had sought medical advice on account of dyspnea I found a double posticus paralysis associated with paresis of the vocal cords. Statistics based on such small

[1] "Arch. de Neurol.," 1897; see "Schm. Jahrb.," vol. CCLVII, p. 82.
[2] "Die laryngealen Störungen bei Tabes dorsalis," Leiden, 1891.

material are, however, of very little value, as were shown by Semon, who found among the 12 first cases of tabes which he examined unilateral or bilateral posticus paralysis five times, whereas the next 30 cases did not yield a single laryngeal disturbance. Of more recent contributions we may mention that of Gerhardt,[1] who found 17 paralyses in 122 tabetic patients, 11 of the posticus (5 bilateral, 4 the right posticus, 2 the left posticus), and 3 of the recurrent laryngeal nerve (1 bilateral, 2 unilateral on the right side). The 3 remaining cases consisted of paralysis of the posticus and thyroid muscles once, paralysis of the recurrent nerve of one side and of the posticus nerve of the other side once, and 2 paralyses of the thyroid arytenoid muscle. In 2 cases there were ataxic movements of the vocal cords; in 4 cases there were laryngeal crises.[2]

Among 100 cases of tabes Semon found 8 unilateral posticus paralyses, 3 bilateral posticus paralyses, and 3 unilateral paralyses of the recurrent nerve.

The most frequent laryngeal complications consist in motor palsies of the laryngeal muscles. The typical tabetic palsy is that of the crico-arytenoideus posticus, either of one or of both sides. In Berger's table of 71 cases of tabetic laryngeal paralysis published up to 1891, there are 33 cases of unilateral paralysis of the posticus, in a few of which there was a coexistent paralysis of the internus; the remaining 38 cases consisted of unilateral paralysis of the posticus, while a few cases showed paralysis of the posticus on one side and paralysis of the recurrent nerve on the other.

From this it would appear that bilateral paralysis of the posticus is almost as frequent as the unilateral form. It must, however, be remembered that the symptoms due to the various forms of paralysis may either be so marked as to produce a very noticeable alteration in the voice or respiration, and thus arouse a suspicion of laryngeal disturbance, or they may be so mild as to escape the examiner's notice altogether, unless every tabetic patient is systematically subjected to a laryngoscopic examination. Hence, unilateral paralysis of the posticus, which does not affect phonation and respiration, is frequently overlooked, while bilateral paralysis of the abductors of the glottis never

[1] Nothnagel's "Spec. Path. u. Ther.," vol. XIII, p. 55.
[2] "Heymann's Handb.," vol. I, p. 705.

escapes detection, because it is always associated with hoarseness and dyspnea.

Complete paralysis of the recurrent nerve is extremely rare in tabes dorsalis. As we have previously stated, a subacute disease affecting the nuclei of the vagus and of the recurrent nerve first produces paralysis of the posticus, which only becomes converted into paralysis of the recurrent later in the disease. The question naturally suggests itself, Why do we not observe this transition from the median to the cadaveric position in those cases of tabes dorsalis which persist for many years, and which, as we know from the reports of autopsies, attack the nuclei in the medulla oblongata? The only clinical fact which points to a progressive nature of posticus paralysis is the occurrence of paresis of the internus, which manifests itself in the laryngeal image in relaxation of the vocal cord, and clinically in the hoarseness and a diminution of the dyspnea due to the bilateral paralysis; the rare cases of recurrent paralysis in tabes, being imperfectly described, are open to question, and can not be regarded as secondary to posticus paralysis. One thing is absolutely certain—the adductors or closers of the glottis are never affected alone in tabes dorsalis. The cricothyroid muscles are also practically never attacked; Gerhardt's case of paralysis of the cricothyroid associated with that of the posticus is the only one that we have met with.[1]

The laryngeal palsies are usually observed in the earlier stages of tabes dorsalis and sometimes precede all other symptoms.

It has been occasionally stated that intermittent paralysis of the vocal cords may be observed in tabetic patients, and that a posticus paralysis may disappear after a few days and return after the lapse of weeks; but the statement has not been satisfactorily proven, and until we have more accurate observations we must assume that once the tabetic paralysis has developed in the larynx there is no hope of cure. The paralysis may, however, develop very gradually, and several cases have been reported which remained constantly under observation and in which a complete posticus paralysis developed in the course of weeks or months: at first there was some power of abducting the vocal cords;

[1] "Ann. des mal. de l'oreille," 1891, p. 480.

this gradually diminished, and finally the vocal cords remained immovable in the median position.

The subjective symptoms are the same as those which occur in paralysis of the vocal cords from other causes. When there is hoarseness, a posticus paralysis produces no symptoms unless the vocal cords are implicated; any marked disturbances always tend to posticus paralysis. The symptoms consist in dyspnea, the voice being only slightly, if at all, affected. As the paralysis develops very gradually, the patient becomes accustomed to the stenotic condition of the rima glottidis, and the interference with respiration is comparatively slight, except during bodily exertion and phonation; during sleep, however, the stenosis becomes very marked. There is a good deal of inspiratory dyspnea, showing itself in loud, sighing inspirations, while the expiration is quite free. There is, of course, a constant danger of asphyxia whenever a greater demand is made on the respiration during any form of bodily activity, so that sooner or later tracheotomy becomes necessary in cases of posticus paralysis.

An experiment performed by Ruault deserves mention in this place. He excised 1.5 cm. from the recurrent nerve in a tabetic patient who was suffering from intense dyspnea due to posticus paralysis, in the hope of bringing the vocal cords into the cadaveric position, but the operation was not followed by any change either in the laryngeal image or in the subjective symptoms of the patient. This is the only case of its kind, and has no particular value.

Ataxia of the vocal cords is a name given to a condition in which the vocal cords execute irregular movements during phonation and deep respiration. Krause was the first to remark that the vocal cords tended to move in jerks and to stop midway between complete adduction and the inspiratory position, producing interrupted or scanning speech. It has been elaborately proved by Burger that this motor anomaly, which occurs exclusively in tabes, is a true ataxia, or disturbance in the coordination of all the antagonistic groups of muscles the cooperation of which is necessary to produce all the movements of the vocal cords.

Laryngeal crises consist in convulsive attacks of cough and dyspnea, and occur in the beginning of, or during the course of tabes, like gastric crises. They differ from attacks of simple laryngeal spasm in that all the other respiratory

muscles are involved. The attacks either occur without any ascertainable cause or after slight external, mechanical, or psychic irritation, particularly swallowing and the introduction of a probe into the throat. According to Oppenheim, pressure on the throat at a point near the anterior border of the sternomastoid muscle at the level of the cricoid cartilage produced attacks of coughing. The attacks occur with variable frequency; they may be repeated several times within a few hours, or a single attack may be followed by a period of freedom lasting for months or years, or may never be repeated. They are usually preceded by a feeling of tickling or burning in the throat; this is followed by a choking attack, with loud, strident inspirations and short, puffing expirations, accompanied by a violent, barking cough which has been compared to whooping-cough. The patient becomes intensely excited and greatly terrified at the idea of impending suffocation, until, after a short time—the attacks rarely last longer than a minute—the respiration is suddenly or gradually restored, sometimes after the expectoration of a little mucus (Burger). They usually end in recovery in spite of their intensity, although Burger was able to collect five cases which terminated fatally during the attack.

Pharyngeal crises are described by Oppenheim as attacks of convulsive gulping movements, which, however, are foreign to our subject. Sensory disturbances of the larynx during tabes are rare. A few cases of anesthesia and hyperesthesia of the pharyngeal and laryngeal mucous membrane have been observed. With regard to the appearances produced by tabes dorsalis in the organ of hearing, I shall here reprint a paper which I read before the Deutsche Otologische Gesellschaft in Dresden, in 1897, and which appeared in a rather inaccessible portion of the reports of that meeting:

In spite of the fact that several papers have appeared on the subject of aural disturbances in tabes dorsalis, opinions are still divided as to their nature, and there are those who deny the occurrence of deafness as a result of tabes.

I shall omit the list of reported cases and shall not repeat the various opinions which have been expressed on this subject, contenting myself with referring to Burger,

Treitel,[1] and Haug,[2] who have given a complete bibliography of the subject. I shall make it my task to attempt to explain the probable nature of ear disease in tabes dorsalis by means of our anatomic and clinical knowledge of the conditions. Although there are no anatomic investigations at my disposal, I shall utilize the results of examinations made on the ears of 27 tabetic patients by a 'Doctor-and' in the Medicinischen Universitäts-Poliklinik at Leipzig. Among these patients there were two cases of impaired hearing which could with certainty be referred to tabes—at least, with as much certainty as the present state of aural examination will permit. I give the percentage as 7.3, although I am reluctant to compute a rate on such a limited number of cases. At least, these investigations show that tabetic ear disease is extremely rare, and tally almost perfectly with the statistics published by Voigt and Treitel, who found auditory disturbances in 2 cases out of 100, and in 2 cases out of 20, or 2% and 10%, respectively. I was unable to obtain the statistics by Marie and Walton in the original, but I have nothing to criticize in the finding of Ménierè's symptom-complex in 17 out of 24 cases; on the other hand, I object strongly to Morpurgo's statement that he found in 43 cases out of 53 auditory disturbances which could be traced to tabes dorsalis—a percentage of 81.13. As the diagnosis was based purely on a positive Rinne test, at reduced hearing-distance, and on a normal condition of the ear-drum, while the air douche was not followed by improvement in the hearing, these statistics are manifestly defective, and after examining the cases I claim that the list does not contain a single case of authentic tabetic deafness.

The infrequency of auditory disturbances in tabes is confirmed by the observation of clinicians with a large amount of material at their command. If we compare the meager reports of deafness with the great number of case histories of tabes dorsalis contained in the literature (we need only mention Erb's statistics of more than 700 cases), our faith in an author who gives a percentage of 81.13 is very much shaken.

The clinical picture of the ear affection is variously de-

[1] "Zeitschr. f. Ohr.," XX.

[2] "Die Krankheiten des Ohres in ihren Beziehungen zu den Allgemeinerkrankungen." Vienna and Leipzig, 1893.

scribed. According to some, the disease presents the characteristics of a lesion in the sound-perceiving apparatus, and is distinguished by otitis interna and by the fact that perception for the higher notes is relatively good, while the hearing is impaired for the deeper and middle notes of the register. Others distinguish two clinical forms, one of which must be regarded as a simple tabetic atrophy of the auditory nerve, the other as syphilitic disease of the labyrinth. The former is gradual in its onset and goes on slowly to complete deafness, being accompanied with tinnitus aurium, but never with vertigo; the latter makes its appearance suddenly, like a stroke of apoplexy, with the phenomena of Ménière's symptom-complex, and in many cases rapidly leads to total deafness.

It follows from this divergence in the conception of the clinical course of the auditory disturbance in tabes that the most various attempts were made to explain the nature of the disease. Some incline to regard the process as an atrophy of the auditory nerve, others attribute the disease to trophic disturbances in the middle ear due to tabetic disease of the trifacial nerve, while a third faction describes the disease as syphilitic. As I shall presently show, all these theories lack the support of anatomic or clinical findings, which alone afford a reliable basis for the description of the disease.

Most authors interpret tabetic disease of the ears as an atrophy of the auditory nerve with the symptoms of a lesion of the sound-perceiving apparatus. It is a proof of our present inability to make a clinical diagnosis of atrophy of the auditory nerve that attempts are constantly being made to discover some minute changes which should be characteristic of tabetic disease of the auditory nerve.

Gradenigo considers it characteristic of tabes when the perception of high notes is relatively good and the loss of hearing applies chiefly to the lower and middle notes; but this phenomenon is not constant, to say the least, for in Habermann's case perception of the lower notes remained good after the patient was unable to hear higher ones. Again, many authors have emphasized the great electric irritability of the auditory nerve, but this phenomenon has not met with universal recognition, and is, moreover, of little value, in view of our imperfect knowledge of the physiology of the electric reaction of the auditory nerve.

The conception of a progressive atrophy of the auditory nerve fails to find pathologic support, because those cases in which disease of the nerve-endings in the labyrinth and in the nuclei was found associated with atrophy of the auditory nerve can not be regarded as cases of primary atrophy of the auditory nerve.

Although on theoretic grounds there may be no objection to this interpretation, since disease of the trunks of the cranial nerves is said to occur in tabes, there is, as I have said, a complete absence of anatomic or clinical proof of its representing the type of a tabetic auditory disturbance; consequently, other explanations were sought. When Lucae was able to refer the impairment of hearing in two tabetic patients to simple disease of the middle ear, furnishing anatomic proof in one case, it gave rise to the opinion that the middle-ear affection was due to tabetic disease of the trifacial nerve, in support of which was cited the fact, determined by the experiments of Baratoux, Gellé, and Berthold, that trophic disturbances may appear in the middle ear after the destruction of the roots of the trifacial nerve. But there is no proof whatever that such an effect on the middle ear through the trifacial nerve takes place in tabes, for in Lucae's case there were no other disturbances, such as would necessarily be present in any disease of the trunk or nucleus of the trifacial, nor was there any anatomic proof of such disease. On the other hand, this explanation is untenable from the fact that Oppenheim [1] found the hearing to be quite normal in a case of marked alteration of the trifacial where the diagnosis rested on an anatomic basis; nor is there any mention of auditory disturbances in another similar case of Oppenheim's. If the opinion that the fifth nerve plays an important part in tabetic deafness were correct, the symptom would certainly have been present in these two cases; its absence, however, makes the hypothesis very improbable.

The syphilitic form of aural disease remains to be discussed. This is particularly insisted upon by Haug,[2] who appears to believe that these cases possess very characteristic clinical features, consisting principally of Ménière's symptoms, with abrupt onset, marked vertigo, vomiting, and sudden deafness, sometimes associated with violent

[1] "Arch. für Psychistrie und Nervenheilk.," xx, p. 147.
[2] *Loc. cit.*

pains. The objective signs, he says, differ from those in a simple case of tabes by the fact that bone conduction is completely abolished. I can not see how these symptoms justify Haug in believing "that he has in all probability to deal with syphilis," since other nervous affections of the ear are accompanied by the same symptoms. Haug also cites the report of an autopsy which he says confirms his opinion, but it is left to the reader to pick out what he considers characteristic of syphilis. It appears that Haug's diagnosis was determined by a little round-celled infiltration which was found surrounding some of the smaller vessels, and the proliferation in the intima of the same. Haug is welcome to consider the ear affection in his case as syphilitic; but, if so, it is not tabetic, and has developed independently of tabes. The explanation that "a primary syphilitic infection may give rise to the combination of lues and tabes which occasionally appears in the organ of hearing in the form of a labyrinth affection" seems rather obscure, and even if we admit a connection between tabes and syphilis, it is, in my opinion, a mistake to look for tertiary syphilitic changes, as such are never found in the parasyphilitic affections, to which the tabetic deafness in this case would belong.

The latest investigations hardly admit of any other explanation of tabes than that it is a disease of the neurons, consisting principally in a lesion of the systems that take their origin in the spinal ganglia. I would apply the same explanation to the auditory disturbances which occur in tabes, and shall, therefore, continue the discussion of this question by referring to the reports of autopsies and clinical observations which have hitherto been published.

For the morbid anatomy, I begin by citing a case of Habermann's,[1] in which the disease was limited to the trunk of the auditory nerve and its terminal endings in the labyrinth, while the nuclei remained intact. It is worth mentioning that the atrophy of the fibers of the cochlear nerve was not so great on the right as on the left side. A bundle of nerve-fibers at the apex of the cochlea and several ganglion cells in the terminal portion of the basilar convolution were preserved—a condition which manifested itself clinically in ability on the part of the patient to perceive deeper tones.

[1] "Arch. f. Ohr.," XXXIII, p. 111.

Next, I will mention Gellé's case, which is always quoted in support of the doctrine of middle-ear disease in tabes. In a woman forty-two years of age, the subject of tabes, there was a sclerosis of the mucous membrane of the middle ear, immobility of the ear-drum and of the chain of ossicles, ankylosis of the stapes, and, as a result of these changes,—to quote the common explanation,—a slight atrophy of part of the various portions of the cochlea, including a disturbance of the nerve-endings on the basilar membrane. The nerves in the lamina spiralis, in the vestibules, and in the semicircular canals were not attacked. In view of the atrophy of the nerve-endings on the basilar membrane I question the propriety of regarding this case as one of simple middle-ear disease, and am inclined to look upon it as a primary peripheral disease of the cochlear nerve.

Strümpell has described one case of tabes in which there had been complete bilateral deafness for four years. Microscopic examinations revealed an evident atrophy of the auditory nerves. Nothing is said about the nuclei or the internal ear, although the statement that the "degenerative process, strange to say, disappears in the restiform body" justifies the assumption that if there had been any disease of the auditory nuclei, which are in such close proximity to the restiform body, it would not have escaped the author's notice.

There remain to be mentioned three cases by Haug in two of which the cochlear and vestibular nerves appeared to be completely destroyed; the trunk and nuclei of the auditory nerve, however, were not examined. In the third case the fibers of the cochlear nerve had disappeared and been replaced by connective tissue, the cells of Corti's organs were opaque, the basilar membrane was preserved, while Corti's membrane and the reticular membrane were the seat of membranous adhesions. Unfortunately, the author does not give a detailed description of this very interesting aural condition, "because it would lead him too far afield," so that there is nothing left to discuss but the medulla oblongata, as the trunk of the auditory nerve was not examined. One of the chief nuclei showed only a slight degeneration of the nerve-fibers, while the other was quite normal. The accessory nuclei could not be made out, as they appeared to be replaced by round-celled infiltration. There was a diminution in the number of fibers in

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the ascending limb and in the mesial root of the auditory nerve, but none in the lateral root.

This is the case that Haug described as syphilitic, and, interesting as it is in certain details, its value is very limited, as the anatomic examination of the medulla oblongata is so imperfectly described that it is impossible to obtain a clear mental picture. The same may be said of Oppenheim's case, in which it is said that nothing definite could be made out in the nuclei of the auditory nerve, although it was quite evident that a large proportion of the root-fibers, which leave the acoustic nucleus at the point where it disappears beneath the nucleus of the vagus and pass up over the ascending root of the fifth nerve, were atrophied. The author also says that "the root-fibers of the auditory nerve are deep red in color, and under a high power appear to have lost the characteristic appearance of nerve-fibers and to be converted into a wavy mass of connective tissue very rich in cells." The trunk of the auditory nerve and the internal ear were not examined.

If we review these cases, we find an evident involvement of the terminations of the cochlear and vestibular nerves in Habermann's and in two of Haug's cases. Gellé's case presents atrophy of the peripheral endings of the cochlear nerve; in Strümpell's case, where the internal ear was not examined, there was atrophy of the auditory nerve, while the nuclei were probably intact, and the condition in the labyrinth and cochlea is not known; finally, in Haug's third case there was evidently deep-seated disease of the auditory nerve and its terminations, associated possibly with disease of the acoustic nuclei. It thus appears that, of all the parts which make up the auditory pathway, the terminations of the cochlear nerve and its ganglion spirale, the vestibular nerve, and the trunk of the auditory nerve are those most constantly affected, while in regard to disease of the nuclei there are no reliable observations. This need not surprise us, if we consider the difficulties which are encountered, even in the normal condition, in the description of the higher auditory pathways.

To make the matter a little clearer, I shall review once more the course of the auditory pathway, and for this purpose shall use the description given by Edinger. The cochlear nerve represents the central process of the cells of the ganglion spirale. From this ganglion, which is situated

in the cochlea, are derived the minute peripheral branches which ramify among the auditory cells. The fibers of the cochlear nerve, which were formerly designated as the posterior roots, enter the ventral nucleus of the auditory nerve, except a small portion which go to the tuberculum acusticum. These structures together represent the primary neuron of the auditory pathway, the ganglion spirale representing the ganglion cells; the cochlear nerve, the axis-cylinder; and the crista acustica, the peripheral terminations. In the vestibular nerve the conditions are not quite so clear; it appears, however, that the ganglia of the primary neuron are situated in the labyrinth, and from that point send out the peripheral terminations to the specialized epithelium in the ampulla, while the axis-cylinder is represented by the vestibular nerve which ends in the dorsal auditory nucleus. The course of the higher pathways, which are regarded as secondary and tertiary neurons, does not interest us in this connection; but we may refer to the developmental relationship probably existing between the ganglion spirale and the spinal ganglia, according to which it is assumed that the ganglion cells of the cochlear nerve in the development of the organism have moved out toward the periphery in a manner analogous to the formation of a spinal ganglion. This relationship appears to me to furnish the explanation for tabetic auditory affections, as I agree with v. Leyden and others in regarding tabes as a disease of the spino-cutaneous sensory neurons. The theory is not so far fetched as would appear at first sight, if we consider the behavior of the peripheral sensory nerves as studied by v. Leyden and his followers and by Oppenheim. Atrophic processes in the peripheral endings of the sensory nerves have been demonstrated in tabes, and, quite recently, Moxter suggested that disease of the optic nerve represents an affection of the neurons beginning in the peripheral endings in the layer of ganglion cells of the retina. As I have already pointed out, the most frequent anatomic alterations in tabetic ear disease are found in the peripheral endings of the auditory nerve, and I accordingly venture to express the opinion that the tabetic ear disease represents a morbid process localized in the peripheral endings in the primary neuron of the auditory nerve; that is to say, in the endings of the cochlear nerve, the ganglion spirale and its axis-cylinder, and the trunk of the auditory nerve.

This conception of the seat of the disease would seem to agree with the clinical picture described by Haug in a pure tabetic form of disease of the auditory nerve, for he described it as a very gradual impairment of the hearing, depending on atrophy, and associated with subjective noises.

As an illustration of the clinical picture, I shall describe one of the two cases of tabetic ear disease which I had the opportunity to observe. A woman fifty years of age had been suffering from symptoms of tabes for a number of years; the patellar reflexes were entirely abolished, the gait was ataxic, the pupils were almost rigid, and Romberg's phenomenon could be easily demonstrated. Within the last three years the hearing, which had never been good, gradually deteriorated. The optic nerve was intact. The patient complained of humming and buzzing noises in the ears, at times so marked as to give the impression of the ringing of bells, the twittering of birds, etc. On examining the ears, an old perforation was found in the right ear-drum in the anterior inferior quadrant, together with marked calcification, while on the left side the membrane showed opacities.

There had evidently existed an old purulent otitis media, which not improbably bore some relation to an atrophic rhinitis still present.

When the functional test was applied, it was found that the internal as well as the middle ear was involved. Bone conduction was entirely abolished. On the other hand, the deeper and middle notes were very faintly heard on the right side (C only in strong vibrations of the fork, $C_2 = -60''$),[1] and somewhat better on the left side ($C = -35$, $C = -5''$); whereas the highest notes (C_5 and Galton's whistle) were quite inaudible on the right side; on the left, however, C_5 could just be heard when it was lightly touched, while Galton's whistle was also inaudible. On both sides speech could be heard only when it was very loud. The occurrence within the last three years of a very noticeable and rapidly increasing deafness justifies the assumption that two different processes are present, one of which ran its course years ago in the middle ear while,

[1] Normal period of tone-perception is as follows:

$C = 110''$.
$C^2 = 70''$.
$C^5 = 15''$.

the nervous deafness of the last few years bears a causal relationship to the tabes.

It remains to speak of the other form of tabetic ear disease which is characterized by abrupt onset and the presence of Ménière's symptoms. Instead of giving a detailed description of its symptomatology, I shall illustrate it by the following history—that of my second case: The patient, forty-eight years old, was suddenly seized during the night with violent vertigo and tinnitus aurium. This was in 1887. He described the subjective noises as "the thundering of a hundred cannons, the ringing of bells, and the rumbling of railroad trains." At the same time he was seized with violent vomiting, repeated from twenty to thirty times before the next morning, when the physician was called and administered a remedy. Immediately after the attack the patient noticed that he was deaf on the right side. Vertigo appeared repeatedly during the next two years, especially after exertion. Tinnitus aurium did not occur again, but for several years he complained of frequent ringing of bells. This now, however, has ceased.

The tabes seems to have appeared at the same time as the aural affection; at least, the patient dates the first occurrence of violent tearing and dragging pains in the legs from that time. Three years ago a visual disturbance was added, consisting, on the left in a complete, and on the right side in a fairly well advanced, gray atrophy of the optic nerve (examination by Professor Schröter). Both ear-drums were normal.

The result of the functional test was as follows: Bone conduction is very much abridged, but the tuning-fork is heard at the point where it is applied, except that when placed on the right mastoid process the sound is heard on the left side. Rinne on the left side, +; the right side could not be tested.

The right ear is entirely deaf for all the notes and also for loud speech, while in the left ear the hearing is normal, except that there is a slight shortening of the period of tone-perception for C.

Similar histories are reported by Althaus [1] and Haug, and it is quite evident that in this form of tabetic ear disease we must look for another situation than the one we have

[1] "Arch. f. klin. Med.," XXIII, p. 601.

been able to demonstrate for the atrophy which begins gradually at the periphery. I shall not attempt to attribute this form to syphilis, as there is not the shadow of a proof that it is of a syphilitic nature. But I do venture the suggestion that this form of tabetic disease of the auditory nerve is localized in the nuclei of the medulla—an assumption which finds some justification in Haug's third case, in which these nuclei were evidently the seat of pathologic changes, while the clinical picture during life had been such as we have just described.

Since it has been found, according to v. Leyden, that the nuclei of most of the cranial nerves are diseased in tabes, either with or without a coexistent degeneration of the corresponding peripheral trunks, I see no reason why we should not assume the occurrence of a similar disease in the auditory nerve. Thus, without being obliged to resort to some other unknown factor, we have a most natural explanation for this form of aural disease. I should like to add another feature, which irresistibly forces itself on the observer's notice in the clinical picture of this last-named variety. It is the abrupt onset of the auditory disturbance which so strongly suggests laryngeal and gastric crises. As these conditions are occasionally found associated with atrophy of the nuclei and roots of the vagus, it seems permissible to assume disease of the acoustic nucleus as the cause of these auditory crises. Finally, the immediate occurrence of paralysis of the vocal cords after laryngeal crises also suggests the probability that an auditory crisis is followed by deafness.

But this leads us into the realm of hypothesis. It must, however, be admitted that such apoplectic forms of deafness not rarely occur in other diseases, so that the question whether they represent an intercurrent affection or a true complication of the primary disease is an extremely difficult one to decide.

Multiple Sclerosis.

In multiple cerebrospinal sclerosis various motor disturbances occur in the larynx which are accompanied with intention tremors and differ from the tremulous movements of the vocal cords due to other causes by the fact that they are observed only during phonation, instead of both in phonation and in respiration.

The most important disturbances are:

1. A retardation of the muscular movements, so that the intended movements of the vocal cords are delayed and accompanied with tremulous movements.

2. Abnormal tendency to fatigue in the muscles. The voice is quickly fatigued by speaking, and it becomes impossible to sustain a tone for any length of time; the speech is scanning and frequently interrupted by high-pitched, explosive sounds, due to the twitching movements of the vocal cords.

3. The tension and adduction of the vocal cords are incomplete, so that the voice is often rough, deep, and hoarse.

4. Muscular palsies. These are rare, and occur more frequently in the adductors than in the abductors of the vocal cords.

To illustrate these phenomena I may mention Löri's [1] observations, in which there was a marked interval between the muscular act of bringing the vocal cords into the phonatory position and the production of the tone. Whenever the patient was asked to imitate a sound, "a slight vibratory motion was immediately observed in the vocal cords, resembling fibrillar twitchings," but adduction and tone-production were delayed longer than in a healthy subject. Von Krzywicki [2] gives this description of the process: During phonation there is a slight twitching in the neighborhood of the vocal processes in the direction of the median line; this soon passes into a general tremor of both cords, which are finally brought together by an abrupt movement; at the end of phonation the return to the respiratory position is accompanied by two or three pendulum-like vibrations toward the median line.

The adductor palsies reported by Löri [3] and Krause,[4] consisting in gaping of the rima glottidis during phonation, are probably to be attributed exclusively to muscular weakness. Riegel's case of paralysis of the recurrent on the right side, with posticus paralysis on the left, is the only one of its kind; it may possibly be due to paralysis of the medullary nucleus.

[1] "Die durch anderweitige Erkrankungen bedingten," etc., p. 12.
[2] "Berlin. laryng. Gesellsch." in "Semon's Centralbl.," VIII, p. 506.
[3] "Deutsche med. Wochen.," 1893, p. 678.
[4] Krause, "Berlin. klin. Wochen.," 1886, p. 557.

Our knowledge of disturbances in the organ of hearing in multiple sclerosis is very imperfect. Moos [1] quotes cases of tinnitus aurium and deafness from the literature, and adds one of his own, in which there was difficult hearing with loss of bone-conduction, associated with anesthesia of both trifacial nerves and ataxia—a condition which led him to seek the seat of the disease in the medulla oblongata. In a case reported by Hess [2] deafness suddenly developed in both ears two weeks after the appearance of palsies in the extremities; the hearing subsequently improved on the left side, but was permanently abolished on the right. Microscopic examination later revealed a sclerotic focus, which had completely destroyed the "nucleus acusticus medius sinister," while on the right side only a moderate number of diseased ganglion cells were found.

Moos is therefore led to believe that the auditory disturbances in multiple sclerosis depend on sclerotic degeneration of the auditory nuclei and of the trunk of the auditory nerve.

We may also have auditory disturbances due to paralysis of the nucleus in epileptiform attacks during the course of a disseminated sclerosis. Oppenheim [3] observed a sudden onset of paralysis of the facial, auditory, and trifacial nerves of the same side, with symptoms of vertigo; the paralysis subsided in a few weeks and was followed after several months by a sudden hemiataxia, which disappeared in its turn.

DISEASES OF THE MEDULLA OBLONGATA.

Syringomyelia.

In this disease we have, either late or in the initial stages, the appearance of bulbar phenomena, manifesting themselves in motor disturbances in the larynx and in reduced reflex irritability of the posterior pharyngeal wall and of the larynx; there is no record of sensory disturbances in these structures having been observed. Motor disturbances of the uvula do not appear to occur. Schlesinger [4] collected 12 cases of syringomyelia with laryngeal complications,

[1] "Schwartze's Handb. der Ohrenh.," I, p. 507.
[2] Dissert., 1888; quoted by Moos.
[3] Quoted by Leyden-Goldscheider, Nothnagel's "Spec. Path. u. Ther.," X, 2. Th., I. Abth., p. 474.
[4] "Neurolog. Centralblatt," 1894, p. 684.

which had been published up to that time, adding five observations of his own. Since then two other cases have been published by Weintraud.[1]

From these 19 observations it appears that the palsy consists usually in unilateral paralysis of the vocal cord, due to paralysis of the recurrent or, rarely, of the posticus; bilateral paralysis of the recurrent was observed in only four cases.

As sometimes occurs in bulbar palsies, the paralysis of the vocal cords is often combined with palsy and atrophy of the trapezius, a fact which, as we have mentioned before, has been used as an argument for the spinal accessory being the motor nucleus of the larynx. Two cases of this kind, in which paralysis of the spinal accessory was combined with posticus paralysis, are reported by Weintraud.[2]

Progressive Amyotrophic Bulbar Paralysis.

Diseases of the bulbar motor vago-accessory nucleus lead to paralyses which may be unilateral or bilateral and may affect either the posticus or the recurrent nerve. They are found in progressive bulbar paralysis more frequently than in any other bulbar disease, but they can not be said to occur with such regularity as to justify the designation of the disease as "paralysie glosso-labio-laryngée."

Anesthesia of the pharyngeal and laryngeal membrane is not present, as a rule, but the pharyngeal, uvular, and laryngeal reflexes are abolished. Schrötter[3] mentions the occurrence of paresthesia in the throat, variously described as a feeling of dryness or as a sense of pressure.

According to v. Leyden,[4] the auditory and Deiter's nuclei undergo atrophy in bulbar paralysis, forming in this respect an exception to the other sensory nuclei, and perhaps explaining the occasional impairment of hearing, going on to deafness, and the tinnitus aurium which is sometimes observed.

The phenomena produced by progressive amyotrophic bulbar paralysis may also be observed in acute bulbar pal-

[1] "Deutsche Zeitschr. f. Nervenheilk.," v, 1894, p. 383.

[2] *Loc. cit.*

[3] "Vorles. über die Krankh. des Kehlkopfs," p. 382.

[4] Von Leyden and Goldscheider, Nothnagel's "Spec. Path. u. Ther.," x, 2, pp. 686 and 701.

sies, such, for instance, as follow embolism or compression of the medulla by tumors. The latter form, which should be designated compression bulbar paralysis, since it is caused by irritation of the medulla from the pressure of the tumor, manifests itself in ataxic movements of the vocal cords.[1] Lastly, we must mention progressive spinal muscular atrophy and amyotrophic lateral sclerosis, which, by combining with bulbar paralysis, may produce bulbar palsies in the larynx.

In pseudobulbar paralysis, which has its principal seat in the cerebrum, Lannois,[2] Cartaz,[3] and Krause[4] observed adductor palsies. The latter might be regarded as cerebral palsies, did we not know that there are always some diseased foci in the medulla and in the pons, besides the principal focus in the cerebrum. As it is uncertain whether vocal-cord paralyses ever occur in cerebral disease, one should never forget that they may possibly be explained by a simultaneous involvement of the medulla oblongata.

NEUROSES.

Paralysis Agitans.

Paralysis agitans gives rise to motor disturbances in the vocal cords affecting the quality of voice and speech. In the laryngeal image we see twitching movements of the vocal cords, which occur regularly in phonation and usually also in respiration, thereby distinguishing themselves from similar movements observed in multiple sclerosis; thus, Fr. Müller[5] observed the phenomenon constantly when the patient exerted himself during the examination, but found that at other times the vocal cords remained perfectly quiet. The tremors may also affect the epiglottis (Rosenberg) and the uvula.

According to Rosenberg,[6] similar disturbances of the speech occur as in multiple sclerosis. We have the scanning speech, described by Charcot[7] as tremulous and interrupted, like "the speech of an inexperienced rider on a

[1] Compare v. Leyden and Goldscheider, *loc. cit.*, p. 711; and Semon-Heymann's "Handb. der Lar.," I, p. 761.
[2] "Rev. de médece," 1885.
[3] "France médicale," Nov. 17, 1885.
[4] "The Jour. of Laryng. and Rhinol.," 1888, p. 255.
[5] "Charité Ann.," 1887, XII, p. 267.
[6] "Berlin. klin. Wochen.," 1892, p. 771.
[7] Quoted by Fr. Müller.

high-stepping horse," and, as a very conspicuous feature, a sudden change from a high to a low register during speaking, due to the inability to sustain a tone for any length of time; as the vocal cords gradually relax their tension and closure of the glottis becomes imperfect, the voice becomes deeper and rougher.

Although only a few cases[1] of laryngeal involvement in paralysis agitans have been reported, it does not seem to be a very rare occurrence, judging from Schultz's report of five observations of tremors in the vocal cords out of twelve cases in Gerhardt's clinic. As the head tremors themselves lead to disturbances of speech, it is very probable that they often mask the symptoms of motor disturbances in the vocal cords, and are thus the cause of the latter's escaping detection.

Epilepsy.

According to Göttstein,[2] anesthesia of the laryngeal mucous membrane is a constant accompaniment of the epileptic attack, and may occasionally persist for some time afterward. The epileptic cry, which frequently heralds the attack, is accompanied by convulsive movements of the laryngeal muscles, consisting either in twitching of the vocal cords or in spasm of the glottis. Semon[3] attributes these phenomena to a cortical irritation.

It is a well-known fact that an epileptic aura often consists in disagreeable olfactory sensations.

We also hear of auditory auræ, consisting either in impaired hearing and deafness, or in the hearing of subjective noises, which may be so marked as to deserve the name of hallucinations. A few cases have been reported in which epileptic attacks were followed by deafness, shown by the functional test to be of central origin.

In one case the deafness was permanent;[4] in another, recovery occurred "after" employment of the galvanic current.[5]

[1] "Charité Ann.," 1887, p. 267. "Berlin. klin. Wochen.," 1892, p. 771. Schultzen, "Charité Ann.," 1894, XIX.

[2] "Lehrbuch der Kehlkopfkrankheiten."

[3] In Heymann's "Handb. der Laryngol.," vol. I, p. 632.

[4] "Arch. f. Ohr.," XXII, p. 205.

[5] "Arch. f. Ohr.," XIV, p. 134.

Chorea Minor.

The use of the term "chorea laryngis" for disturbances of coordination in the movements of the vocal cords in various diseases is unfortunate, as it confuses the question whether laryngeal disturbances may occur in chorea minor. It seems probable that the vocal cords rarely participate in true choreic movements. Schrötter [1] says that temporary convulsive contractions occur in the larynx simultaneously with similar movements in the respiratory muscles, and represent the cause of the sighing or gasping inspirations, which are frequently audible at some distance.

The disturbances of speech, which in severe cases manifest themselves as sudden interruptions by shrill whistling sounds, are also to be attributed to choreic movements of the muscles concerned in deglutition and respiration.

According to Haug,[2] the tensor veli palati and tensor tympani sometimes share in the contractions, and lead to the production of subjective or objective noises in the ear, described as the cracking of nuts or the crackling of paper.

Hysteria.

Hysteria is to be regarded as a neurosis without anatomic basis in which an alteration in the psychic condition of the patient is the most important factor. The clinical picture may assume an infinite variety of forms, but all the symptoms have this in common—that they affect those functions of the body which are, to a certain extent, subject to the will of the patient. Strümpell insists that hysteria shows so marked a preference for the voluntary functions that it does not occur at all in the domain of the involuntary muscles and of the automatic reflexes. This law appears to hold good for the regions with which we are now concerned, for we shall see that hysteric phenomena, although they may at first sight appear to be quite irregular, nevertheless seem to follow a certain system in the upper air-passages and in the ear, inasmuch as they do not affect those functions of the larynx and of the ear which are considered purely automatic, such as the respiratory dilatation of the glottis and the static function of the ear. It is con-

[1] "Die Krankh. des Kehlkopfes," First Edit., p. 388.
[2] "Die Krankh. des Ohres," etc., p. 204.

venient to divide hysteric disturbances into those which affect the sensory region, those which affect the motor region, and those which affect the regions of special sense.

In the sensory disturbances in the region of the upper air-passages we have to deal with mucous membrane in the nose, with the exception of the vestibule; and in the organ of hearing, both with epidermis (which covers the internal auditory meatus and the external surface of the ear-drum), and with mucous membrane, which forms the lining of the middle ear and of the tubes.

The disturbances of special sense include alterations of the senses of smell and hearing; these are usually associated with sensory disturbances.

The motor disturbances embrace those which occur in the regions of the pharyngeal and laryngeal musculature and in the muscles of the tubes and of the middle ear.

1. In the first group we have anesthesia and hyperesthesia, analgesia and hyperalgesia.

Although in general the law holds good that mucous membranes adjoining the external skin, as in the vestibule of the nose, present the same sensory disturbances as the adjoining external skin, this, as pointed out by Lichtwitz,[1] is not the case when large areas of the surface are diseased. This applies particularly to hemianesthesia of the body-surface. We learn from the accurate studies of Lichtwitz, which harmonize with the results obtained by Thompson and Oppenheim,[2] that in a purely cutaneous hemianesthesia the hemianesthesia of the mucous membrane is never complete, but usually extends over both halves of the body; and, just as there may be cutaneous hemianesthesia without involvement of the mucous membranes, so the latter may be affected while the skin remains intact. A characteristic feature of hysteric disturbances of sensation, which is also observed when other portions of the body are attacked, is that the distribution of the anesthesia, instead of corresponding to the distribution of certain nerves, is a diffuse one, without any reference to the innervation—so much so that it furnishes an important point in the differential diagnosis from anesthesias due to an organic, anatomic lesion in which the sensory disturbances are strictly limited to the domain of the affected nerves.

[1] "Les anesthésies hystériques des muqueuses," etc., Paris, 1887.
[2] "Arch. f. Psych. u. Nervenheilk.," xv.

The nasal mucous membrane is less apt to be included in the anesthesia than are the other mucous membranes of the upper air-passages; the anesthesia never affects its entire surface, and there are always islands of intact mucous membrane between the anesthetic areas. According to Lichtwitz, the septum always escapes, except in the lower anterior portion, which lies within the domain of the vestibule.

The pharyngeal mucous membrane is very frequently affected, perhaps more frequently than any other part of the upper air-passages, including the larynx. The occurrence of anesthesia of the epiglottis, which was regarded by Chairon as pathognomonic of hysteria, is not confirmed by other authors. It is difficult to determine the frequency of anesthesia in the upper air-passages, for it necessarily escapes the notice of the patient and does not betray itself to the physician by any visible alterations; it can, therefore, be detected only by a special examination. Sometimes we are led to suspect it by the ease with which a laryngoscopic examination is performed, for the absence of subjective complaints is often a very marked feature. The fact that particles of food do not find their way into the air-passages and lead to inspiration pneumonia, as in all organic palsies, especially in postdiphtheric anesthesias and in bulbar palsies, leads us to conclude that reflex swallowing and reflex cough are not affected in anesthesias of the pharynx and larynx. The choking reflex, on the other hand, is frequently abolished.

Diminished sensibility of the mucous membrane frequently occurs in hysteria, but it can not be separated from anesthesia.

Analgesiæ have been observed in connection with analgesia of the general body surface; they may or may not be associated with anesthesia.

There is another important group of hyperesthesiæ and paresthesiæ, which differ from the sensory disturbances just described in the fact that they occasion marked subjective symptoms and may lead to demonstrable alterations in the mucous membrane in the form of hyperemias and consecutive chronic catarrh, as a result of the violent efforts at swallowing and the constant coughing and hawking. In such cases the paresthesia is usually caused by the conversion of the temporary irritation in the

larynx or pharynx—a passing inflammation or slight catarrh—into a permanent neurosis, as a result of the hysteric disposition. The patients complain of a persistent tickling sensation, which they usually attribute to a foreign body, like a particle of food, on a particular spot in the throat. In either case a coryza or simple sore-throat is followed by hyperesthesia of the pharynx. Sometimes a nauseating bit of food or a mouthful of foul water, during bathing, for instance, may lead to hysteric disturbances.

Hyperesthesia and paresthesia manifest themselves in the nasal mucous membrane in sneezing, and in the pharynx and larynx in coughing, hawking, and straining, or even in vomiting, or sometimes in a constant desire to swallow. To this category belongs the globus hystericus, which gives the sensation of a spherical body moving up and down between the region of the epigastrium and the throat.

Under the name of anæsthesia dolorosa Schnitzler has described a peculiar variety of sensory disturbance in the pharynx, in which subjective pain in the throat is associated with anesthesia of the soft palate, of the posterior laryngeal wall, and of the larynx.

When the diseased structures were examined, the finding in the nose and throat was negative, while in the pharynx and larynx certain alterations were seen, which were interpreted as a mild congestive or hypertrophic condition. The mucous membrane in the pharynx, and occasionally at the entrance to the larynx, may be abnormally pale, and in that case represents part of a general anemia, such as we expect to see in hysteric women. On the other hand, the opposite condition may be present, in which case the congestion of the mucous membrane must be regarded as a result of the irritative cough, and as an expression of the plethora such as we not infrequently observe in elderly women with excitable sexual feelings that have never been gratified.

The auricle, the skin of the external auditory meatus, and the epidermis of the ear-drum are all subject to the sensory disturbances which have just been mentioned, and which may be either unilateral or bilateral. The anesthesia is not accompanied by any subjective symptoms, for Gellé's opinion that the ability to locate the source of the sound is disturbed in unilateral anesthesia of the ear-drum has not been confirmed. In hyperesthesia both of the external ear

and of the Eustachian tube there are marked subjective symptoms in the form of paresthesia in the external auditory meatus or an aggravation of an already existing trifling affection of the organ of hearing. The presence of very small masses of cerumen on the walls of the external meatus often produces a distressing sense of a foreign body in hysteric persons, while the scratching induced by the irritation of the paresthesia may set up a mild dermatitis, which gives the patients great distress, so that they often complain of a feeling as if there were a movable foreign body or an insect in the ear. The hyperesthesia in the mucous membrane of the tubes is said to manifest itself in unusual sensitiveness to catheterization and to the passing of a bougie. Hyperesthesia and hyperalgesia of the ear occur, being usually localized in the middle ear (otalgia tympanica) or in the mastoid process. The phenomenon known as "*transfert*," to which we shall refer again later, has been observed in the ear during these disturbances of sensibility.

In this connection we must mention the so-called *hysterogenic zones*, irritation of which is said by Lichtwitz to bring on a hysteric attack, unless the parts have been previously cocainized. They have been located in the mucous membrane of the nose, in the larynx, on the posterior wall of the nasopharynx, on the posterior surface of the uvula, on the mucous membrane of the tubes, in the external auditory meatus, and on the ear-drum ; sensation was preserved in the parts affected. These hysterogenic zones possess no great practical value, and are no more significant than any other sensitive portions of the body, the irritation of which, as is well known, may produce hysteric attacks. The fact that a hystero-epileptic attack or any other motor reflex phenomenon may be induced by probing a hypertrophied region in the pharyngeal mucous membrane, by introducing a catheter into the tube, or by syringing the ear for the purpose of removing a plug of cerumen, is of no more significance than the production of similar phenomena by irritation of any given region on the external skin. Thus, touching a small wart on the hand has produced general hysteric convulsions which disappeared after the wart was removed with a galvanic cautery. But this would hardly justify us in speaking of a hysterogenic zone, any more than the phenomenon of a woman being seized with hysteric

respiratory convulsions when a catheter was introduced into the Eustachian tube.

Hysteric disturbances in the nerves of special sense manifest themselves in the olfactory nerve as hyperosmia, hyposmia, and parosmia. The effect on the function of hearing in hysteria shows itself either in deafness (hypæsthesia acustica) or in abnormal sensitiveness of the auditory nerve (hyperæsthesia acustica). These disturbances may occur suddenly after fright or any violent emotion, or they may develop gradually. It is very rarely that they constitute the only hysteric symptom, as other nerves of special sense, particularly the optic nerve, are nearly always involved. Natier claims to have observed a remarkable combination of hysteric deafness with inability to speak, or with functional disturbances of the voice, such as stammering and hoarseness. Diminished or increased sensibility of the auditory nerve are, as a rule, unilateral. In a relatively large number of cases the power of hearing is found to be abnormally increased on one side and diminished on the other. This condition is very conspicuous in a case reported by Urbantschitsch,[1] in which the phenomenon known as "*transfert*" was typically present. As Habermann [2] and others have been able to perform this experiment, which is specially dwelt on by French writers on hysteric deafness, it may be worth while to devote a few words to it in this place.

By means of a small magnet or a piece of metal (gold) placed on the sound ear it is possible to transfer the hysteric deafness or any existing anesthesia of the ear to the sound side. Urbantschitsch observed that the higher notes are the first to be transferred, and, conversely, as the "*transfert*" returns to the side originally affected, the hearing is lost for the higher notes sooner than for the lower ones. A somewhat frequent phenomenon is the alternation between diminished and increased sensibility at certain hours of the day.

The auditory disturbance in hysteria is characterized by a uniform loss of perceptive power for all the notes in the scale. It does not exactly correspond either to a nervous affection or to disease of the sound-perceiving apparatus, for Rinne's test is usually positive, while in Weber's experi-

[1] "Arch. f. Ohr.," vol. XVI, p. 176.
[2] "Prag. med. Wochen.," 1880, No. 22.

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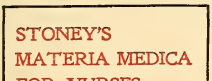

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ment the tuning-fork is heard equally as often on the sound as on the diseased side, or may even not be lateralized to either side.

Subjective noises are not constantly present. Hyperæsthesia acustica manifests itself either in morbidly sensitive hearing or in the appearance of hysteric convulsive phenomena, when certain tones are heard. Steinbrügge [1] describes a very interesting case of this kind, in which the hearing of musical tones was followed by hysteric respiratory convulsions. It is worthy of remark that disturbances of the hearing in hysteria are not accompanied by disturbances of the equilibrium, such as occur in organic disease of the auditory nerve as a part of Ménière's symptom-complex.

The symptoms which we have just described make it appear very improbable that the seat of the lesion is in the auditory pathway. The phenomena of "*transfert*" and the variation in the degree of deafness appear to show that the perceptive faculty of the nervous auditory apparatus is not impaired, and that the disease is to be sought in an interruption of the nervous pathway which conveys the auditory impression to the seat of consciousness and transforms it into a sound; hence, we probably have to deal with a simple disturbance of coordination in the central pathways.

3. Hysteric motor disturbances are purely disturbances of coordination; there is no paralysis, for the motor power is not lost, but there is inability to perform the muscular movement in such a way as to produce the desired effect. This disturbance between the will and the act may consist either in an excessive amount of muscular activity, or in a total want of the necessary movements, so that either the picture of a convulsion or that of a paralysis may be produced. When we speak of a relation between the will and the deed, it is implied that the disturbance can concern only movements which are subject to the will and under the control of cortical centers of coordination, while reflex movements, such as are originated below the region of the will, like the respiratory gaping of the glottis, are not subject to these hysteric disturbances.

We must, however, discuss at somewhat greater length

[1] "Zeitschr. f. Ohr.," XIX, p. 328.

certain hysteric posticus paralyses, as Penzoldt,[1] West,[2] and Dufour[3] have described bilateral hysteric posticus paralyses, and Treupel[4] observed a unilateral paralysis which was associated with paresis of the internus. But as West remarks that the vocal cords were in close contact during inspiration and changed to the cadaveric position during expiration, and as Penzoldt speaks of intense dyspneic attacks, which imply that there were intervals of freedom in which the "paresis" subsided, it does not appear to me that these isolated cases—the rarity of which is in such strong contrast to the frequency of hysteric motor disturbances in general—furnish sufficient proof for a diagnosis of hysteric posticus paralysis. I rather incline to believe that we have to deal in these cases with a so-called perverse action of the vocal cords, a phenomenon which depends on a disturbance of coordination and consists in the approximation of the vocal cords toward the median line in deep inspiration, followed by separation during expiration, such as we very often observe in excitable persons at their first laryngoscopic examination.

Motor disturbances rarely occur in the muscles of the pharynx; the abnormal movements are synchronous with the respiration, and manifest themselves as rhythmic inspiratory contractions of the two posterior pillars of the fauces, usually accompanied by simultaneous contractions of the adductor muscles of the vocal cords. Hysteric motor disturbances in the larynx chiefly affect the muscles concerned in phonation. As we have previously stated, the participation of the abductors in the loss of phonation or a convulsion, as assumed by Przedborski,[5] has not been proved. The most important symptom of these affections is the hysteric aphonia, a simple disturbance of coordination in which there is imperfect coaptation of the adductor muscles and inability to maintain the tension necessary for the voice production. The laryngoscopic image is subject to enormous variations, and we find in hysteria every alteration in the shape of the glottis that can possibly be conceived as produced by the failure in action of indi-

[1] "D. Arch. f. klin. Med.," XIII, p. 118.

[2] See "Semon's Centralbl.," X, p. 39.

[3] "Thèse de Montpellier," Jan. 9, 1891; "Semon's Centralbl.," IX, p. 96.

[4] "Die Bewegungsstörungen im Kehlkopfe bei Hysterischen," Jena, 1895.

[5] "Mon. f. Ohr.," 1885, No. 11.

vidual groups of muscles. Thus, we get the picture of paralysis of the internus, of isolated paralysis of the inter-arytenoideus, of the lateral crico-arytenoid, or of the thyro-arytenoid muscles, or various forms of paralysis combined, so that, for instance, the action of the interarytenoid and of the lateral crico-arytenoid muscles may be in abeyance. These associated palsies of the adductors are particularly characteristic of hysteria and are rarely found in other diseases. That these hysteric disturbances do not correspond to palsies in the ordinary sense of the term is shown by the fact that during the examination the vocal cords often approximate almost to the point of contact at the first attempt at phonation, and then immediately separate and assume a position simulating one of the forms of paralysis mentioned; that is to say, the muscles do not lack the faculty to perform the movements, but there is a psychic inability to perform the desired act. This finds further confirmation in the remarkable phenomenon that hysteric subjects, although incapable of uttering a single word in a loud voice, often phonate or cough during the laryngoscopic examination, or regain their power to speak in their dreams or during hypnosis; while even a greater paradox is presented by those cases in which the singing voice is perfectly preserved while there is complete aphonia.[1] Spasm of the glottis and so-called laryngeal cough are occasionally observed, and are to be regarded as spastic phenomena or hyperkinetic motor neuroses. This hysteric cough and other laryngeal noises (bruits laryngés), which may have the sound of bleating, howling, or grunting, are described by Charcot.[2] Characteristic features are their recurrence at definite hours and their complete cessation during sleep. The cough or other noises may occur only once or be repeated several times in rhythmic order; dyspnea or apnea never occur, no matter how often the attacks may be repeated. There are no objective signs in the larynx or in the lungs, and it is noteworthy that the condition is not accompanied by any other hysteric symptoms, unless there be certain disturbances of sensation and of special sense, such as anesthesia and diminution in the field of vision.

[1] Griffen, see "Semon's Centralbl.," x, p. 312. Gerhardt, "Kehlkopfgeschwülste," etc., Nothnagel's "Spec. Path. u. Ther.," vol. XIII, 2. Th., 2 Abth., p. 50.

[2] "Med. du syst. nerv.," II, p. 443.

All these forms of hysteria occur, either suddenly, under the influence of fright, traumatism, etc., or develop gradually. The nature of hysteria is such that any peculiar movement that has once been executed under the influence of some momentary external agency persists as the effect of a morbid imagination (Treupel). As the disease does not depend on any material alterations, the line of treatment is clearly indicated; an attempt should be made to free the patient from the morbid impression that he is unable to use his voice, and the normal power of the muscles should be restored by systematic exercise of the voice and of the respiration. Moritz-Schmidt[1] divides hysteric palsies into three grades, on a purely external practical basis, as follows:

First, cases in which the voice may have been lost for some time, immediately reappears on laryngoscopic examination, but is lost immediately afterward.

Second, cases in which the voice does not appear during examination and the patient whispers.

Third, cases in which the patient can not even whisper —a condition which has been designated apsithyria.

A single case of motor disturbance in the muscles of the ear, consisting in contractions of the tensor tympani, and giving rise to subjective cracking noises, has been reported.[2] Among other manifestations of hysteria hemorrhages from the ear have been mentioned. As the cases referred chiefly to females, or persons suffering from neurasthenia as the result of masturbation, we refer to the chapter on the sexual organs for these affections.

Certain neurasthenic and hysteric conditions which are produced by traumatism and very frequently give rise to disturbances in the ear are closely allied to hysteria. Observations have been reported in regard to ear disease in "railway spine,"[3] and in regard to traumatic hysteria of the ear[4] [5] after a stroke of lightning, while innumerable hysteric and neurasthenic symptoms following injuries of the head have been reported. The diagnosis often presents the greatest difficulty to the physician on account of

[1] Second Edit., p. 706.
[2] Freund u. Kayser, "Deutsche med. Wochen.," 1891, No. 31.
[3] Baginsky, "Berlin. klin. Wochen.," 1888, No. 3.
[5] Freund u. Kayser, "Deutsche med. Wochen.," 1891, No. 31.
[4] "Arch. f. Ohr.," vol. XXIX, p. 327, and vol. XXXVIII, p. 102.

the want of any definite objective findings. Where no external injury is visible, the patients usually complain of increasing difficulty of hearing and tinnitus aurium. Vertigo, which is usually absent in hysteria, is conspicuous in these cases, but it is not at all clear that the symptom is due to a lesion in the ear itself. The results of the functional test are often the same as in hysteria. It is often extremely difficult to interpret them correctly, both because the patient is in a state of mental excitement and gives contradictory and unreliable answers, and because the deafness can not, as a rule, be ascribed with absolute certainty to the traumatism, as it is a well-known fact that the hearing is very apt to be affected in traumatic neurasthenia with the remains of an old aural disease.

APPENDIX.

NASAL REFLEX NEUROSES.

Since the ground was broken by the works of Hack the doctrine of nasal reflex neuroses has received general recognition, and thus it may be said that another morbid process has been added to the domain of pathology. As, however, Hack in his first publications gave such a liberal interpretation to the concept of nasal reflex neurosis that experienced observers were led to warn the profession against a too general application of his propositions, and other authors after Hack in their uncritical laxity extended the range of reflex neuroses almost indefinitely, it seems well to define what is meant by reflex neuroses and their various forms as determined by the results of accurate experimentation.

Among the reflex conditions which can with certainty be attributed to irritation of the nasal mucous membrane we include sneezing, trifacial cough, spasm of the glottis, and asthma. In addition, there may be some effect on the action of the heart, but here we must take into account the possibility of vasomotor disturbances, such as the so-called vasomotor coryza and "hay fever." Our physiologic investigations of these reflexes are based on the investigations of François Franck which have been utilized more than any others in the following description. The sensory nerves of the nasal cavity are derived from the trifacial. The anterior ethmoidal nerve, a branch of the nasal nerve of the first division, supplies the anterior portion of the nasal cavity corresponding to the external nose, while the remaining portion of the interior of the nose is supplied by the posterior nasal branches of the second division, and by a branch of the dental nerve of the third division.

These nerves transmit the nasal reflexes which are known as sneezing, nasal cough, and reflex spasm of the glottis.

The sneezing reflex may be produced in any part of the

nasal mucous membrane, as any one can convince himself. The anterior and posterior extremities of the middle and inferior turbinals, and the corresponding parts on the septum, are said to constitute a special irritative zone, as the reflex is most easily produced in these regions. The sneezing reflex may also be produced by irritation at some distance, which is transmitted to the nasal cavity through the channels of the trifacial nerve. Everybody is familiar with the production of the nasal reflex by sudden illumination of the eye, such as occurs when we look into the sun, the reflex in this case being carried from the ciliary to the anterior ethmoidal nerve by way of the nasal nerve. The sneezing can usually be prevented by exerting pressure on the trunk of the ethmoidal nerve at a point where it is superficial, as on the inner upper wall of the orbit and at the lower border of the nasal bone, where the external branch leaves the inner surface of the nasal cavity, between the bone and the cartilage. The reflex act of sneezing itself is effected by the respiratory muscles, and consists in a deep inspiration followed by a sudden explosive expiration with the glottis widely gaping and the soft palate shutting off the oral cavity from the nasopharynx, so that the entire respiratory blast escapes through the nose under high pressure.

The "nasal cough," and the reflex convulsions of the glottis and of the bronchi in the form of spasm of the glottis and asthma, represent various grades of a reflex action transmitted through the same channels. The impulse travels toward the center along the channels of the trigeminus, and returns toward the periphery in those of the vagus. François Franck and Lazarus have furnished exact experimental proof of this reflex. By irritating the nasal mucous membranes it is possible to constrict the lumina of the bronchi, but as soon as the vagus is excluded the experiment becomes impossible. The marked contraction of the bronchial muscles may even give rise to visible retraction of the intercostal spaces (François Franck). The spasmodic nature of this reflex from the nasal mucous membrane may manifest itself in spasm of the glottis as well as in asthmatic symptoms. The latter is to be regarded as a combination of all the forms now under discussion, and was observed in animals, which, after irritation of the nasal mucous membrane with the galvanocautery, fell to

the ground in a condition of asphyxia, with respiration arrested either in inspiration or in expiration, and recovered very slowly. Milder grades of the attack showed themselves more in a change of the respiratory rhythm and general restlessness of the animal.

The nasal reflex neuroses manifesting themselves in cardiac affections in the form of retardation of the pulse and cardiac arhythmia have also been proven by direct experimentation. Another group of reflex neuroses, revealing themselves clinically in swelling and redness about the nose and eyelids and in headache and vertigo find their physiologic explanation in the vasomotor disturbances produced in the nose. To this class belong the vasomotor secretory neuroses which are described as vasomotor coryza, hydrorrhea of the nose, and hay fever. Their etiology is usually not ascertainable ; most likely they represent a reflex neurosis which any accidental external factor may induce in hysteric and neurasthenic subjects. It is not as yet generally admitted that hay fever belongs to this group. It is possible that paralysis of the sympathetic may also produce hydrorrhea of the nose, although the hypothesis is not confirmed by experience. In one case of unilateral paralysis of the cervical sympathetic I observed that the erectile tissue in the turbinals was more swollen on the diseased, than on the sound side, but there was no nasal secretion, and the swelling yielded promptly to cocain. Finally, we must include in this group the reflexes transmitted to the sexual apparatus from the nose, since they are to be regarded in the main as the effect of vasomotor irritation.

We distinguish two kinds of vasomotor disturbances of nasal origin, one of which manifests itself in the erectile tissue and mucous membrane of the nose itself, while the other finds expression in the vessels of other organs when the sensory nerves of the nose are irritated. The reflexes of the first variety are chiefly vasodilator in character, such as we see after probing a normal nasal mucous membrane. The reflexes of the second group, on the other hand, present various characters. Thus, François Franck found that irritation of the nasal mucous membrane produced a dilatation in the vessels of the head and a constriction in the superficial and deep vessels of the extremities.

We often see the statement that reflex neuroses may be produced by the sense of smell, and the literature contains

a large number of cases in which the perception of certain odors was followed by reflex conditions. One woman was attacked with sneezing fits whenever she smelled roses; another whenever she was exposed to the foul smells of manure from a horse or cow stable; epileptic and asthmatic attacks, and even reflex irritation of the genitalia through the olfactory nerve, have been described, and the sexual excitement induced by smelling the opposite sex has even been interpreted as a reflex. For the first group effected through the sense of smell we must assume an idiosyncrasy depending on hysteric predisposition, while the second form can be explained on psychologic grounds only. The scenting of the opposite sex evokes not a reflex, but a sensuous excitation analogous to that conveyed through the eye or the ear; instance the call of the male to the female among animals.

When we attempt to explain the production of a change in the respiratory rhythm, which appears to be the effect of reflex irritation through the olfactory nerve, we meet with a greater difficulty. Gaule,[1] however, suggests that the change in the respiratory rhythm is not so much a reflex act of the organism to protect the body against the invasion of deleterious substances, as it is an effort to adapt the respiration to the act of smelling.

It follows from these considerations that the existence of nasal reflex neuroses now rests on a firm theoretical basis, instead of, as formerly, on mere clinical observation consisting chiefly in *post hoc propter hoc* arguments, such as improvement after local treatment of the nose. But to be quite exact, we should in addition demand an absolute clinical proof for all nasal reflexes, viz., that they can only be produced from the nasal mucous membrane, that they may be completely arrested by anesthetizing the membranes, and that they can only be finally cured by direct treatment of the offending spot in the nasal mucous membrane. These points should be particularly insisted upon in the diagnosis of all doubtful cases.

Hack uttered the opinion in his first publication that the reflexes can only be produced from certain definite regions in the nasal mucous membrane corresponding to the position of the erectile tissue. He set up the hypothesis that

[1] Heymann's "Handb. der Laryng.," vol. III.

the irritation of the sensory nerve endings of the nasal mucous membrane is secondary to the swelling and engorgement of the erectile tissue, so that the reflex is not due to the primary cause but to the irritation of the nerve endings by the swollen erectile tissue. This theory was antagonized by Fränkel and others, and it has now practically passed into oblivion. Yet it appears to throw some light on certain doubtful points, for, as pointed out by Hack, it is a well-known fact that the reflexes are less likely to be produced by chronic catarrhal conditions associated with great hyperplastic swelling than they are by the milder hyperemic processes, inducing an intermittent swelling of the nasal mucous membranes which would be more likely to irritate the nerve endings.

But we can dispense with this artificial theory of Hack's by laying down the maxim that reflex neuroses are most apt to occur when opposing regions of the mucous membrane periodically come into contact with one another, nasal respiration, being still intact.

This intermittent contact is lost when the adjoining regions are brought into constant apposition by conditions of hyperplasia or by the formation of large polypi. Such an irritation is possible in any part of the nasal mucous membrane where the lateral wall is capable of touching the median wall, and it is not necessary to limit its predilection to the region of the erectile tissue. It is true that contact will occur most frequently between the inferior and middle turbinate bones, where the embedded erectile tissue on one side impinges on the other, on the tubercle of the septum, a condition favorable for the development of such hyperemia.

The individual shape of the interior of the nose also plays an important rôle. Thus a marked deviation, or a spine or crest on the septum approaching the lateral nasal wall favors a periodic contact between the opposed mucous membranes even when the swelling is very slight.

In the etiology of nasal reflex neuroses we must not neglect those conditions in which slight hyperplastic processes are found at the anterior extremity of the middle turbinate bones without any other pathologic conditions in the nose, or the presence of small nasal polypi just beginning to grow from the middle turbinate in the middle meatus. In this case the reflex neuroses appear

to be produced by contact of the hyperplastic mucous membrane with the free border of the inferior turbinated bone.

Another etiologic factor in the production of nasal reflex neuroses is said to be found in adhesions between adjoining portions of the nasal mucous membrane and in distortions and overstretching of the membrane by the contraction of cicatricial tissue. It is also quite conceivable that the continued presence of a foreign body in the nose might lead to reflex neuroses by irritation of the sensory nerves.

An important rôle in the production of the clinical picture which we are considering must be conceded to suppurations within the nose originating in adjacent cavities. We must mention in particular suppuration in the antrum, especially in those cases where, owing to a marked alteration in the region of the middle meatus, there is only a slender stream of pus in the middle meatus to indicate the disease. In these cases the nerve endings in the mucous membrane are irritated by the pus which enters through the nasal orifice of the respective cavity and, by moistening the surrounding mucous membrane, materially affects its nutritive conditions, as is shown by the polypoid hypertrophies produced in the later stages of the disease.

Finally, we must emphasize that a nervous disposition is necessary for the production of the nasal reflex. The nasal mucous membrane is in a condition of abnormal excitability in which a mild irritation, such as in the healthy subject would produce only a slight swelling of the nose, is capable of evoking a whole complex of reflex phenomena. While the pungent odor of certain substances, such as flowers or agricultural products or the inhalation of smoke and dust-laden air, produces in a healthy man only the normal reflexes, consisting in swelling of the mucous membrane, increased secretion, and the act of sneezing, the same influences in the hypersensitive mucous membrane of hysteric and neurasthenic persons suffice for the development of pathologic reflexes manifesting themselves in cough, asthma, or even in the symptom-complex of hay fever.

It is worth mentioning that pertussis has also been regarded as a reflex neurosis derived from the nasal mucous membrane, and it is said that the attacks can be considerably mitigated by cocainizing that structure.

In connection with the nasal reflex neuroses we must

refer to certain conditions which are usually included among them, but really only represent the sequelæ of interference with nasal respiration; they are not the effect of reflex irritation, but are produced mechanically by interference with nasal respiration and the secondary changes in the organism. In these conditions we do not have to deal with a neurosis which can be shown to follow irritation of certain regions of the nasal mucous membrane, nor with a neurosis which can be suppressed by cocainizing the respective regions in the nasal mucous membrane. They represent rather the expression of insufficient respiration and defective oxidation of the blood, and may present themselves under a great variety of forms.

It is hardly necessary to say that we must carefully guard against too liberal a construction of the significance of nasal stenosis. The most frequent manifestation is that known as aprosexia, which can be seen characteristically in children suffering from adenoid vegetations. Enuresis nocturna, chorea, and epilepsy have also been included among the ultimate effects of nasal stenosis, but great care should be enjoined in interpreting such cases, remembering that very often a few accurate observations are obscured and vitiated by subsequent carelessly reported cases, and thus the whole doctrine discredited.

Under the name of aprosexia (ἀπροσέχειν τὸν νοῦν) Guye [1] has described a clinical picture consisting of inability to fix the attention on one subject, of unusual forgetfulness manifesting itself in the rapid disappearance of mental impressions which originally were acquired only at the expense of great effort, and finally of headache, which in some cases was limited to a feeling of constant or intermittent pressure in the head, while in others it produced all the phenomena of violent hemicrania, especially during the morning hours.

According to Guye, we should distinguish three varieties of aprosexia, the first of which is physiologic and the effect of overexerting the brain; the second, neurasthenic, as a consequence of pathologic brain fatigue; while the third represents the nasal aprosexia, now under discussion.

It arises in consequence of nasal stenosis associated with swelling and stasis in the venous and lymphatic channels of

[1] "Deutsche med. Wochen.," 1887, No. 43, and 1888, No. 40.

the nasal mucous membranes. The pathogenesis of aprosexia is readily understood when we consider the intimate relation existing between the lymphatic spaces and blood-vessels of the nasal mucous membrane and the subarachnoid space. Schwalbe and Retzius were able to inject the lymphatic vessels of the nasal mucous membranes through the arachnoid space. An equally intimate relation exists between certain venous regions of the nose and the interior of the skull, although in this case the blood stream which, according to Zuckerkandl, is directed brainward, does not suggest stasis in the intercranial venous channels so much as an engorgement of the nasal veins with stagnant venous blood containing a large percentage of CO_2.

Enuresis nocturna occurs with comparative frequency in children suffering from obstruction of the nose due to adenoid vegetation or other causes. Grönbech,[1] however, believes that the cases are probably due to a certain disposition to enuresis, since adenoid vegetations are very common, and the combination of enuresis with nasal obstruction ought therefore to be observed much more frequently.

The most familiar, and at the same time most plausible hypothesis, is that the relation between the two diseases depends on an excessive amount of CO_2 in the blood due to defective respiration, as a result of which there is a mild degree of carbonic acid poisoning, which in turn leads to relaxation of the vesicle sphincters.

A simpler explanation is that the enuresis is due to the fact that the children are restless and only half asleep in consequence of the defective respiration. It is often found in connection with pavor nocturnus (terror infantium) in mouth-breathers, and is explained by the fact that sleep is frequently interrupted by cessation of the breathing due to reflex closure of the mouth.

In rare cases, choreic movements, especially of the face, —as, for instance, wrinkling of the brow or twitching of the corners of the mouth,—are described. Tic convulsif and epileptiform conditions have been attributed to nasal obstruction. Although we usually find the note that removal of the cause of the nasal stenosis, such as adenoid vegetation or hypertrophies of the mucous mem-

[1] "Arch. f. Laryng.," II, p. 224.

brane, was followed by cessation or diminution of these "reflex conditions," the cases can not be accepted without a reservation, as the causal relationship between nasal stenosis and such convulsions is still very obscure.

Occasionally examination of the nose or other minor nasal operation is followed by a partial epileptic attack of syncope similar to its epileptic equivalent, by sudden excitement, or by temporary unconsciousness; such convulsive attacks are not to be regarded as reflex phenomena, they are the product of a violent psychic irritation in subjects of a neurasthenic or hysteric disposition. I have, however, seen a true epileptic attack produced in an epileptic subject by endonasal interference.

THE SIGNIFICANCE OF SOME OF THE CRANIAL NERVES IN RHINOLOGY AND OTOLOGY.

The Trifacial Nerve.

The trifacial nerve is the sensory nerve of the mucous membrane of the nose and of its accessory cavities, and is therefore involved in any diseases affecting these structures. Hence nasal diseases are frequently accompanied by neuralgia and reflex phenomena conveyed through the branches of this nerve. The nasal reflexes have been mentioned, and in speaking of diseases of the eye it has been said that irritation of the anterior ethmoidal nerve and of the nasal branches of the second division of the trifacial in the interior of the nose may give rise to reflex epiphora. We will now consider exclusively the sequelæ which take the form of neuralgia.

Supra-orbital neuralgia is a frequent symptom of disease of the frontal sinuses. Both acute rhinitis with inflammation of the mucous membrane in the accessory cavity, and acute or chronic suppuration of the frontal sinuses may lead to a typical neuralgia of the first division of the fifth nerve. The implication of the nerve finds a general explanation in the fact that branches of the supra-orbital extend to the anterior and lower wall of the frontal sinus, and may thus transmit the pain of an inflammation to the trunk of the nerve; but, in addition, certain individual anatomic conditions play an important rôle, since the distance of the supra-orbital nerve from the walls of the cavity varies with the dimensions of the frontal sinus.

The symptoms of a neuralgia secondary to disease of the frontal sinus are the same as those of simple neuralgia. Points of tenderness are found at the supra-orbital foramen and at the inner upper angle of the orbit where the ethmoidal nerve leaves that cavity. There is tenderness of variable degree at the anterior lower wall of the frontal sinus, while epiphora and slight edema of the upper eyelid occur, with lancinating, often periodic pains radiating toward the forehead and occiput. These symptoms may be either unilateral or bilateral, depending on the nature of the primary disease.

When the neuralgic symptoms predominate in the clinical picture, a diagnosis is usually impossible without a nasal examination until circumscribed edema and bulging of the orbital or anterior wall of the frontal sinus make their appearance, when even the general practitioner who is not versed in rhinology can no longer entertain a doubt of the existence of suppuration in the frontal sinus. It can not be denied, however, that even the rhinologic examination is not always absolutely clear, as simple catarrhal changes of the nasal mucous membrane may occur in primary trifacial neuralgia as the effect of a reflex vasomotor and trophic disturbance. The nasal condition establishes the diagnosis when the neuralgic pains are associated with discharge of pus from the middle meatus or with hypertrophic or polypoid changes in the neighborhood of the hiatus semilunaris and on the middle turbinated bone.

Neuralgia of the infra-orbital nerve may occur in connection with disease of the antrum of Highmore. The canal for the transmission of the nerve fills the upper (orbital) wall of the cavity and projects sharply into the lumen, while its numerous dental branches course along the inner surface of the lateral wall in minute grooves covered only by the mucous membrane of the sinus. Although the infra-orbital nerve is nearer to the antrum of Highmore than is the supra-orbital nerve to the frontal sinus, neuralgia of the inferior orbital nerve is rarer than supra-orbital neuralgia. This is perhaps explained by the drainage conditions of the cavity and the location of the nerve at the roof, while in the frontal sinus the retention of pus frequently leads to such an increase of the pressure as to cause bulging of the walls of the cavity. This increase of pressure rarely occurs in empyema of the antrum—disregarding

cysts and tumors which give rise to different appearances,—as the communication between the cavity and the nose, although situated somewhat high and affording incomplete drainage, is still much more free than that of the frontal sinus, which is situated in the narrow infundibulum of the hiatus semilunaris. Hence pressure on the nerve canal or on the nerve itself, if the canal is gaping, is not likely to occur. But if there is marked retention, the pressure manifests itself chiefly on the floor rather than against the orbital wall, and a bulging of the lateral wall of the cavity is much more likely to occur as it is in part membranous and corresponds to the lateral wall of the nose.

As the dental branches from their superficial position are more exposed to disease, the inflammation may spread from them to the nerve-trunk and give rise to typical neuralgia, or at least to tenderness at the point of exit of the infra-orbital nerve.

Grünwald[1] states that the sphenopalatine ganglion, on account of its close proximity to the anterior and inferior walls of the sphenoidal sinus and the ethmoidal cell, is liable to become involved in caries of these bony cavities.

In neuralgia of the first division there is a point of tenderness on the external nose where the external branch of the anterior ethmoidal nerve passes out between the nasal bone and the lateral cartilage to the skin covering the tip of the nose.

During operations on the septum and on the nasal floor, the pain often radiates to the upper incisors and to the anterior part of the hard palate, owing to the distribution of the terminal branches of the nasopalatine nerve of Scarpi which leaves the nasal cavity through the incisor foramen to reach the oral cavity.

The relations between the trigeminal nerve and the organ of hearing consist in :

(1) Disturbances in the ear in disease of the trigeminus and its branches; and,

(2) Disturbances of the trigeminus in diseases of the ear.

The organ of hearing receives sensory nerves from the trifacial, the auricle and external auditory meatus being supplied by the auriculotemporal nerve, a branch of the

[1] "Die Eiterungen der Nase," 2d ed., p. 125.

third division, while another branch running from the small superficial petrosal to the tympanic plexus along the median wall of the tympanum effects a connection between the trifacial and the tympanic plexus by means of the otic ganglion.

Otalgia is a favorite but very misleading term for all kinds of earache. It is, of course, convenient, and as it is a very general expression, it does not commit one to anything, so that it is often used to describe any obscure symptom. It would be well, however, to use the expression otalgia as a diagnostic term only when it is synonymous with neuralgia otitica or neuralgia tympanica, affections which point to implication of the trifacial and of its aural branches.

These neuralgias occur most frequently in caries of the upper or lower molars. Körner [1] gives as a symptom of this form of otalgia which is often difficult to distinguish from toothache: an increase of the pain in the ear when the gland between the lower jaw and the hyoid bone is pressed upon.

Similar pains are complained of in diseases of the articulation of the jaw which are variously described as rheumatism (Schwartze[2]), or neuralgia (Bruck[3]). From my own observation of a similar case I know how difficult it is to interpret the earache correctly, and it may often be impossible to determine whether there is an otalgia due to radiation of the pain through the branches of the trifacial, or whether the pain in the joint is erroneously referred to the ear.

The distribution of the trifacial is also responsible for the radiation to the ear of pains which have their origin in the nasopharynx, the pillars of the fauces, the lateral wall of the pharynx, and the base of the tongue. Neuralgic pains in the ear occurring in connection with disease of the epiglottis and of the larynx are probably transmitted by the pneumogastric through its auricular branch, which is one of the sensory nerves of the external ear.

It would take too long to enumerate all the diseases capable of producing neuralgia of the ear in this way. The commonest of them are ulcerations, acute inflammations, angina (especially tonsillar abscess), and inflammation and

[1] "Zeitschr. f. Ohr.," XXX, p. 133.
[2] "Die chir. Krankh. des Ohres."
[3] "Deutsche med. Wochen.," 1895, No. 33.

swelling of the base of the tongue and of the epiglottis. Finally, "otalgia" has long been known as a characteristic sign of carcinoma of the larynx.

In trifacial neuralgia the pain frequently radiates to the ears, or may even be especially marked in the auriculotemporal nerve. Krepuska [1] met with a case of primary sarcoma of the Gasserian ganglion which began with obstinate neuralgia. Lesions of the nucleus or trunk of the trifacial nerve produce anesthesia of the external auditory meatus and of the auricle. The eruptions of herpes zoster which occasionally become localized in the auricle may perhaps also be referred to this innervation.

As the tensor tympani muscle is innervated by a motor branch of the trifacial which is given off from the otic ganglion, we should expect to find interference with this muscle in disease of the trifacial nerve. It is, however, very difficult to prove that such is actually the case, as we possess no reliable means of distinguishing the functions of the internal muscles of the ear. The functional disturbances which result are very slight, for paralysis of the muscle does not affect the general power of hearing; it only induces hyperesthesia to very high tones, while a contraction of the muscle diminishes the vibrations of the ossicles and increases the pressure in the labyrinth. Clonic spasms of the tensor tympani, in which the tensor veli palati usually participates, manifest themselves as cracking noises in the ear; they were first described by Schwartze.[2]

Extirpation of the Gasserian ganglion or of the second or third divisions of the trifacial nerve, now quite frequently done for therapeutic purposes, should afford us a means of studying the function of the muscle, but the observations in this respect have so far been very disappointing. In the case published by Krause, [3] the hearing was not affected unfavorably by extirpation of the Gasserian ganglion, and in the case reported by Aster [4] from Czerny's clinic, of resection of the second and third division of the fifth nerve, the hearing remained intact for three weeks after the operation, when another complication occurred which will be referred to later. The only phenomenon

[1] Krepuska, "Zeitschr. f. Ohr.," XXX, p. 189.
[2] "Arch. f. Ohr.," II, 4.
[3] "Münch. med. Wochen.," 1895, Nos. 26 and 27.
[4] "Beitr. z. klin. Chir.," XI, 3 Heft.

that may perhaps be regarded as the result of paralysis of the tensor tympani is that described in one of Krause's cases. The patient complained of a peculiar sensation in the temporal region which she compared to the ticking of a watch, and which, she said, she had never observed before the operation. But as this phenomenon is rather a symptom of clonic muscular cramp or irritation of the trifacial than of a paralysis, it is difficult to establish any causal relationship between it and the extirpation of the nerve. Moos [1] believes that the hyperesthesia of the trifacial nerve may, without the motor branches being involved, lead to auditory disturbances in the form of abnormal sensitiveness to certain kinds of tones and noises, and explains the phenomenon as due to an increased tactile sensibility of the external auditory meatus to the unusual sound waves! Whether the tinnitis aurium which accompanies toothache is due to reflex muscular irritation or to vasomotor influences has not as yet been determined. Schwartze [2] explains it as a reflex irritation of the auditory nerve through the trifacial, but the explanation does not seem very clear to me.

Urbantschitsch[3] reports a series of observations which have been interpreted as the effect of reflex irritation of the sense of hearing from various regions supplied by the trifacial nerve.

The conditions found after extirpation of the Gasserian ganglion fail to confirm the observation of various authors, based on experimentation, that lesion or division of the trunk of the trifacial nerve sets up an inflammatory process in the mucous membrane of the tympanum, designated by Berthold [4] [5] as otitis media neuroparalytica. The latter authority claims that lesions of the trifacial, either in its continuity or in the roots, may produce all stages of inflammation in the middle ear from simple vascularization to suppuration, and Baratoux [6] found that this was confirmed by his experiments; while Kirchner, [7] after dividing the inferior dental nerve in a cat, and subjecting it to

[1] "Virch. Arch.," vol. LXVIII.
[2] "Berlin. klin. Wochen.," 1866, Nos. 12 and 13.
[3] "Lehrb.," p. 349.
[4] "Schwartze's Handb.," p. 315.
[5] "Zeitschr. f. Ohr," X.
[6] "Arch. f. Ohr.," XIX, p. 199, 200.
[7] "Mon. f. Ohr.," 1882, No. 4, and comp. "Arch. f. Ohr.," XX, p. 58.

electrical irritation, observed a more marked dilatation of the vessels in the tympanic cavity and increased secretory activity of the mucous membrane. We may disregard those cases in which toothache was followed by acute exudative middle-ear catarrh (Walb), or those in which paralysis of the trifacial was followed by more or less complete deafness, as the connection between the two diseases is not clearly shown. In fact, the opposite appears to be proved by Krause's cases, in which extirpation of the trifacial had no effect on the power of hearing. Asher's case can not, I think, be used in the evidence; a serous exudation in the middle ear associated with chronic catarrh of the nasopharnyx developed three weeks after resection of the second and third divisions of the trifacial nerve.

As neuralgia of the ear may be due to diseases of the trigeminal nerve, conversely this nerve may become implicated in diseases of the ear. The trunk of the trigeminal nerve may suffer in endocranial complications of middle-ear disease, in pachymeningitis, in extradural abscess, and in serous and purulent meningitis. Phlebitis of the cavernous sinus gives rise to neuralgia of the first division of the fifth nerve (Körner [1]).

As the Gasserian ganglion is situated in Meckel's recess, on the upper surface of the petrosal portion of the temporal bone, a purulent otitis media may, by extension toward the apex of the petrous portion, lead to marked nutritive disturbances, involve the Gasserian ganglion and produce trifacial neuralgia, as was first described by v. Tröltsch [2] and Schwartze,[3] and later by Habermann.

Chorda Tympani.

Lesion of the chorda tympani gives rise to disturbances in the sense of taste in the anterior two-thirds of the tongue, and as the nerve passes through the tympanum [4] and is exposed to injury by any pathologic process present in that cavity, it deserves special mention.

The chorda tympani is given off from the facial nerve a

[1] "Otit. Hirnerkrankungen," 2d edit., 1896, p. 67.

[2] "Arch. f. Ohr.," IV, p. 126.

[3] "Arch. f. Ohr.," XIII, p. 110.

[4] Comp. v. Frankl-Hochwart, Nothnagel's "Spec. Path. u. Therap.," vol. XI, II, Th. 4. Abth. "Die nervösen Erkrankungen des Geschmacks," etc., the literature will be found best in Urbantschitsch, "Lehrb. der Ohrenheilk." and in "Schwartze's Handb.," I, p. 468.

little above the point where the latter leaves the tympanum. It reaches this cavity through a special opening in its bony wall, and after sweeping from behind forward and upward between the long process of the incus and the handle of the malleus, partially covered by the posterior ventricular fold, passes through the Glaserian fissure to reach the base of the brain, and is continued from that point to the lingual nerve of the third branch of the trifacial, with which it becomes united.

Although a branch of the facial, it really belongs to the trigeminus, from which it is originally given off, and only accompanies the facial for a short distance. As it is proven that injury of the facial above the geniculate ganglion has no effect on the sense of taste, the fibers of the chorda tympani may leave the facial either by way of the great, or by way of the small superficial petrosal nerve. But as this point is still in dispute, opinions are divided as to whether the chorda tympani belongs to the second or to the third division of the trifacial; if to the former, the nerve runs from the great superficial petrosal nerve through the Vidian to the sphenopalatine ganglion; if to the latter, from the small superficial petrosal nerve to the otic ganglion.

The innervation is even more complicated, and the symptom of loss of taste at the tip of the tongue becomes more obscure, when we remember that the path of the fibers of taste is a variable one; they may pass directly from the chorda tympani to the otic ganglion without passing through the facial nerve, or they may join the facial after its exit from the stylomastoid foramen without utilizing the chorda tympani. The first mode of distribution may be inferred when a lesion of the facial in the temporal bone between the geniculate ganglion and the region of the chorda tympani does not affect the sense of taste; the latter, if a lesion of the facial, external to the stylomastoid foramen, is followed by loss of the sense of taste.

The conditions being thus inconstant, we can not wonder that the functional disturbances which occur after destruction of the chorda tympani during its course through the middle ear do not tally with our expectations. The statements of a patient are of no value in the determination of the frequency with which disturbances of the sense of taste occur in middle-ear disease when the chorda tympani is destroyed, for we know by experience that such disturbances

may often escape detection even in those who are given to observing themselves most carefully. Thus Carl,[1] who had suffered from purulent otitis media for many years, was very much astonished when he one day discovered that he had entirely lost the sense of taste in the anterior portion of the tongue; and the investigations of Urbantschitsch,[2] who made a careful examination of 50 patients suffering from middle-ear diseases—mostly chronic suppurations,—and found that 46 were suffering from a gustatory disturbance, go to show that, unless a special examination has been made with a view to determine the presence of such a disturbance, case histories are of no value in determining the frequency of its occurrence. Carl observed sharp stinging sensations on the left margin of the tongue, beginning at about the middle and shooting to the tip with lightning rapidity, thus corresponding to the distribution of the chorda tympani; the pain occurred whenever he cleaned his ear with cotton pledgets or irrigated it with astringents and salicylic acid.

While we are on the subject of gustatory disturbances due to diseases in the middle ear, we must not omit to mention those which occur after injury of the tympanic plexus in the distribution of the glossopharyngeal nerve.

The tympanic plexus is formed by the terminal branches of Jacobson's nerve, a branch of the glossopharyngeal, and connects with the trifacial and facial nerves and with the sympathetic caroticotympanic plexus. Owing to the connection of Jacobson's nerve, which is given off from the petrosal ganglion, with the small superficial petrosal nerve, this plexus contains both gustatory fibers from the petrosal ganglion through the glossopharyngeal nerve, and gustatory fibers from the otic ganglion through the trifacial, so that one would expect disturbances of the sense of taste after lesions of the tympanic plexus, and they have in fact been lately reported by Schlichtling[3] from Körner's clinic.

According to Urbantschitsch and others,[4] the secretion of saliva may be affected by chemic or mechanical irritation of the tympanic plexus and of the chorda tympani either

[1] "Arch. f. Ohr.," X, p. 163.

[2] "Anomalien des Geschmack," Stuttgart, 1876 (from quotations in Urbantschitsch's "Lehrb. der Ohrenheilk." and elsewhere).

[3] "Zeitschr. f. Ohr.," XXXII, p. 388.

[4] "Schwartze's Handb.," I, p. 471.

during medication (alum, salicylic acid, etc.), or during instrumental treatment (probing), or by inflammations of the nerves in purulent otitis media, as the nerves which supply the parotid gland are derived from the sympathetic and glossopharyngeal, while those which supply the other salivary glands are found in the chorda tympani.

Facial Nerve.

As the facial and auditory nerves are united in their course as far as the internal auditory meatus, they are often attacked by the same disease. Hence the combination of central facial paralysis with nervous auditory disturbance may afford a valuable hint for the localization of an endocranial lesion.

The facial nerve has other important relations with the organ of hearing, inasmuch as it is the motor nerve for the muscles of the ear.

The muscles of the auricle and the stapedius muscle are supplied by the facial nerve, and their function is therefore impaired in any paralysis of the facial situated centrally, or originating in the respective muscular branches.

Paralysis of the posterior auricular nerve, which supplies the occipital muscle and the retrahens, attrahens, and attollens aurem muscles, manifests itself in immobility of the auricle, and according to Erb [1] may indicate whether the seat of the paralysis is above or below the region of this nerve, which leaves the facial after its exit from the stylomastoid foramen. Patients suffering with facial paralysis frequently complain of tinnitus aurium and difficult hearing. As the stapedius muscle is supplied by the facial nerve, the symptom is usually referred to paralysis of the nerve, and Asher [2] saw this confirmed anatomically in a case of facial paralysis due to direct pressure of a cerebral tumor in the occurrence of atrophy of the stapedius muscle.

According to Göttstein,[3] Hitzig was the first to point out that patients suffering from peripheral facial paralysis experience a loud buzzing noise in the ear whenever they attempt to move the paralyzed half of the face, because the voluntary impulse, being unable to innervate the muscles of the face, expends all its force on the branch to the sta-

[1] "Arch. f. klin. Med.," XV, p. 22.
[2] "Zeitschr. f. klin. Med.," vol. XXVII.
[3] "Arch. f. Ohr.," XVI, p. 61.

pedius muscle which is still intact. In a number of Erb's cases, and occasionally in the latest literature, these patients complain of certain auditory disturbances which they describe as hyperacousis (oxyocoia), tinnitus aurium, and heightened electrical irritability of the auditory nerve.

It is, however, more than doubtful whether all these phenomena can be referred to the stapedius muscle, and we can not lose sight of the possibility that the auditory nerve itself may be involved on account of its proximity to the facial.

Buzzing noises in the ear on the paralyzed side in peripheral facial paralyses are probably always to be referred to abnormal activity of the stapedius muscle.

Hyperacousis or oxyocoia can be referred to the loss of function of the stapedius muscle which, according to Lucae,[1] accommodates the ear to the highest known musical tones, and paralysis of which is followed by abnormal perceptive power for deep notes and increased sensitiveness to all musical tones and similar sounds, particularly for deep tones, so that if the noises are at all loud they may produce a sensation of pain.

In regard to increase in the electrical irritability of the auditory nerve, there is an observation by Seterblad[2] which is often quoted, but has never been confirmed by anybody else.

We may also mention another aural symptom in facial paralysis; it is the prodromal pain in or behind the auricle which, according to Oppenheim,[3] may appear even when the paralysis does not originate in the ear.

The facial nerve, from its close proximity to the auditory and its passage through the temporal bone, comes into very close relations with the organ of hearing. It accompanies the auditory nerve from its exit at the medulla oblongata as far as the internal auditory meatus; hence facial paralyses due to lesion of this portion of the nerve-trunk are frequently accompanied by auditory nerve deafness. Among the causes which may produce such lesions, tumors and aneurysms at the base of the brain, basal meningitis, syphilitic pachymeningitis, and gummata are the most frequent.

[1] "Berlin. klin. Wochen.," 1874.
[2] "Zeitschr. f. Ohr.," XVI, 292.
[3] "Lehrb. der Nervenkrankh."

After leaving the auditory nerve in the inner meatus, the facial continues its course in the Fallopian canal, and at the geniculate ganglion turns backward and downward, crossing the posterior portion of the median wall of the tympanic cavity, and finally, after passing downward along the floor of the posterior wall of the external auditory meatus, leaves the skull through the stylomastoid foramen.

During its course through the petrous portion of the temporal bone the nerve is well protected, and is therefore little exposed to diseases other than tumors and traumatic fractures of the bone. Hence such a paralysis is an important sign of disease in the internal ear. The nerve is most exposed to disease during its passage through the middle ear.

Facial nerve palsies are often observed in acute inflammations of the middle ear, and are explained by extension of the inflammation either through the openings which exist in the canal of the facial nerve for the passage of the nerve to the stapedius and the chorda tympani, or through congenital clefts which not infrequently expose the nerve at different points in the middle ear. It has also been stated [1] that a facial paralysis may be caused by inflammatory hyperemia in the domain of the stylomastoid artery, which supplies both the tympanic cavity and the auditory nerve. The danger to the nerve is of course enormously increased if the suppuration in the middle ear is associated with carious disease of the bone, as such a complication leads to sequestration of the bony wall of the facial canal. Injury of the facial during operations can be avoided if the surgeon possesses any knowledge of the anatomic relations of the nerve and even a moderate experience in operative technic. Nevertheless, they are seen only too frequently after extirpation of the petrous portion of the temporal bone and radical operations.

DISEASES OF THE MENINGES AND OF THE CEREBRAL SINUSES.

Their Significance in Connection with the Nose, Larynx, and Ears.

Diseases of the meninges may involve the cranial nerves and thereby produce pathologic conditions in the organs under discussion.

[1] Schwartze, "Die chirurg. Krankh. des Ohres," p. 174.

Such changes have been observed in pachy- and lepto-meningitis and in tubercular and syphilitic meningitis, and recently the opinion is becoming more and more prevalent that serous meningitis is often responsible for palsies of the cranial nerves. Thus, paralysis of the vocal cords has been observed in various diseases: in epidemic cerebrospinal meningitis irritative conditions in the muscles of the larynx may occur, as observed by Oppenheim (quoted by Kraus), along with irregular twitchings in the lower distribution of the facial nerve, in the uvula, and in the vocal cords, taking the form of continuous rhythmic and isochronous contractions in the vocal cords. Occasionally olfactory disturbances are reported as signs of involvement of the olfactory nerve, but the most frequent sequelæ of diseases of the meninges are found in lesions of the auditory nerve or of the labyrinth. Hence, in any case of greatly diminished hearing or deafness acquired in early youth, we should always take into account the possibility of an antecedent inflammation of the meninges if there is no history of an infectious disease.

The aural disturbance may originate in disease either of the auditory nerve or of the labyrinth, since it is well known that the sheath of the auditory nerve and the aqueducts of the vestibule and of the cochlea present a natural pathway for the spread of the disease from the interior of the cranium to the internal ear.

In cases where it is doubtful whether the seat of the aural disturbance is to be sought in the trunk of the auditory nerve or in the labyrinth, a coexistent facial paralysis may point to the localization of the lesion in that part of the trunk of the acusticus which lies in close proximity to the seventh nerve.

Among the diseases of the meninges acute cerebrospinal meningitis plays a very important rôle, and I take up the consideration of this disease now rather than among the infectious diseases, because it gives rise for the most part to the same varieties of secondary disease of the cranial nerves as a meningitis due to other causes.

We learn from studies on the etiology of epidemic cerebrospinal meningitis that the nose plays an important part in the genesis of the disease, the meningococcus intercellularis (Weichselbaum) being constantly found in the nose and its accessory cavities. Although the significance of

this bacteriologic finding is somewhat weakened by the fact that Schiff[1] found virulent cocci in 4 out of 28 cases of persons who were not suffering with epidemic cerebrospinal meningitis, it is nevertheless probable that infection very frequently takes place through the nose, because the disease often begins with coryza (Strümpell); and we have Weigert's[2] authority for the statement that catarrhal inflammations are frequently found in the accessory cavities of the nose at the autopsy.

It is quite possible that the ear as well as the nose may in some cases afford entrance to the pathogenic microorganisms of acute cerebrospinal meningitis. The meningococcus intercellularis has, indeed, been found in isolated cases in the aural secretion,[3] but not with sufficient frequency to warrant a general conclusion as to its primary significance in the production of a secondary meningitis. As pointed out by Leyden[4] and Schwabach,[5] purulent otitis media occasionally coexists with the general disease, so that the thought naturally suggests itself that both affections are produced by the same pathogenic microorganism. Schwabach was able to prove in one case, in which the internal auditory meatus and dura mater were found to be entirely free from pus at the autopsy, that the suppuration of the middle ear was not a secondary inflammation due to extension from the cerebrum.

Purulent otitis media is, however, comparatively rare as a complication of acute cerebrospinal meningitis, and far less frequent than the other form of the disease which is due to direct extension of the purulent process from the meninges to the internal ear.

It has been proved by numerous anatomic investigations[6] that the inflammation extends either along the sheath of the acusticus or through the aqueducts of the labyrinth where the purulent or hemorrhagic inflammatory process is followed by extensive tissue-destruction. As we have just stated, the suppurative process in the middle ear often begins in the first stage of the systemic disease; the deafness which must be attributed either to suppuration within

[1] "Centralbl. f. inn. Med.," 1898, No. 22.
[2] "Deutsche Arch. f. klin. Med.," XXX.
[3] Fromann, "Congr. f. inn. Med.," 1897.
[4] Nothnagel's "Spec. Path. u. Therap.," vol. X.
[5] "Zeitschr. f. klin. Med.," XVIII.
[6] Comp. Moos, "Schwartze's Handb.," I, p. 575.

the labyrinth or to disease of the nerve-trunk occurs either in the course of the disease or as a sequel.

The impairment of hearing, which is often accompanied with vertigo and vomiting, symptoms due probably to implication of the vestibular segment of the labyrinth, presents no definite characteristic, but usually goes on *pari passu* with the rapid extension of the alterations in the middle ear and labyrinth, and attains a very high grade in a few days. Often it goes on to total deafness, affecting one or both ears, and may even render the patient deaf and dumb, because the ravages of the disease are usually so great that the power of hearing can not be restored. The statistics in deaf and dumb asylums present convincing proof of the prominent part taken by acute cerebrospinal meningitis in the medical history of their inmates.

Diseases of the Meninges in Nasal Affections.

There have been reported in the literature a small number of cases in which disease of the meninges followed disease of the nose and of its accessory cavities. The cases have been collected by Grünwald [1] and Dreyfuss,[2] the most frequent diseases being purulent meningitis, cerebral abscess, and thrombosis in a sinus, especially in the cavernous sinus. The number is, however, very small, and the cases lack uniformity. Hence it will be impossible to show the existence of a definite relationship, as will be seen to be the case in otitic cerebral diseases. Therefore it is not altogether Dreyfuss' fault that he failed in his attempt to give a systematic presentation of "diseases of the cerebrum and its adnexa following suppurations in the nose," [3] in spite of his perseverance and industry in looking up all the literature bearing on the subject.

The interior of the cranium may become infected either from the nose or from its adjacent cavities. In the former case, infection is transmitted by the lymphatic and vascular channels, which, as we have repeatedly stated heretofore, establish an intimate relationship between the upper segment of the nasal cavity and the anterior fossa of the cerebrum. Thus all kinds of inflammations, including the reactive form due to the use of the galvanic cautery, and infectious diseases of the upper portion of the nose,

[1] "Die Lehre von den Naseneiterungen," Munich, 1896, p. 125.
[2] Jena. Fischer, 1896. [3] *Loc. cit.*

which approximately corresponds to the ethmoid bone, frequently set up an irritative process in the meninges and lead to grave constitutional phenomena. Considering the frequency of galvanocaustic interference, the cases that go on to a purulent meningitis are, however, comparatively rare. The latter complication is particularly to be dreaded after tamponade of the upper portion of the nose on account of the resulting retention of secretion, which is always of an infectious nature. The fissures which are said to be occasionally present in the cribriform plate of the ethmoid bone are, according to Dreyfuss,[1] of some significance in the genesis of rhinitic cerebral complications, but the cases of Chiari and Kaiser, on which he bases his theory, did not appear to furnish a satisfactory proof, and it is difficult to believe that "the unfortunate subjects of this anomaly are in danger of contracting meningitis after any ordinary coryza," and that "even a violent blowing of the nose is fraught with great danger in such individuals" (Dreyfuss).

The second mode of infection of the cerebrum, from the accessory cavities of the nose, follows caries of the walls of the cavities, a frequent sequel of chronic suppuration. The danger of infection to the brain from the diseased cavities necessarily depends on their anatomic relations with respect to the interior of the cranium and the thickness of their walls, as in some cases of chronic suppuration with caries several cavities are affected at the same time, so that it is often impossible to determine the exact spot from which the suppuration has extended to the cerebrum. The possibility of such an etiologic connection must be considered in all diseases of the meninges in patients who are the subjects of chronic suppuration from the nose. The frontal sinus, the ethmoidal cells, and the sphenoidal sinus represent the cavities which are in direct relation with the base of the skull, and which therefore constitute a more or less serious menace to the cerebrum according to the thickness of their walls.

When, as a consequence of caries, there are evident defects in these walls through which the pus can find entrance into the interior of the skull, the mode of infection is manifest, but there are other cases of purulent meningitis in which, as I have myself seen, the path followed by

[1] Jena. Fischer, 1896, p. 47.

the pus in traveling from the accessory cavities to the serous membranes can not be demonstrated postmortem, the bony wall being apparently intact, so that an osteophlebitis must be assumed to explain the infection of the meninges. It would appear from the reported cases, including my own, that the sphenoid sinus is most apt to transmit the infection, in spite of the thickness of its roof, which corresponds with the sella turcica.

Diseases of the Meninges and of the Cerebral Sinuses in Ear Disease.

The importance of aural disease in the production of secondary diseases of the meninges and of the cerebral sinuses can not be overestimated, and the doctrine of otitic cerebral disease now forms one of the most important chapters of otology.

Since Schwartze introduced operative measures in the treatment of ear diseases, since the progress of brain surgery removed all obstacles in the way of opening the skull, since cerebral localization became more and more perfected, so that after exposing a morbid focus in the temporal bone the extension of the process to the interior of the cranium could be observed clinically, the great significance of acute and especially of chronic suppurations from the middle ear, with accompanying caries of the bone and cholesteatoma formation in the production of secondary cerebral disease, has won general appreciation. A large proportion of all brain abscesses—estimated at one-third—are secondary to disease of the middle ear, the infection having been carried by means of the diseased meninges. Most cases of convex meningitis, of extradural abscess, and of diseases of the cerebral sinuses are to be referred to aural disease, and the most practical proof of the frequency of otitic cerebral complications is found in the great activity of the aural surgeons, who, with untiring energy, publish all their operative cases. The great number of analogous cases has made it possible to determine the pathogenesis and symptoms of these complications. Körner in "Otitic Diseases of the Brain, the Meninges, and the Blood-vessels," [1] has reduced this doctrine to a system, and this has recently been added to very largely by the important contributions of Jansen.

[1] Weisbaden, Bergmann, 2d edit., 1896.

The scope of the present volume does not permit an adequate description of the significance of these important relations. That belongs to the domain of special text-books on otology, and I shall content myself with pointing out the channels by which suppurations in the middle ear may reach the interior of the cranium, and by describing the most important clinical pictures.

I already touched upon the question of the causal disease in cerebral complications when I said that they may be produced either by acute or by chronic disease of the ear. Among the acute suppurations from the middle ear, the most dangerous are those which follow acute infectious diseases, such as scarlatina, diphtheria, typhoid, and influenza, and to these we must add the diseases of the bone which often follow acute inflammation in the course of diabetes mellitus and tuberculosis, and which from the rapidity of their course may reach the interior of the cranium in a few weeks. As a chronic otitis media is in danger of spreading to the cerebrum, the caution can not be too often repeated that removal of a chronic suppuration, whose destructive effect on the bone can not be controlled, is the first law in the treatment of ear diseases. If all ordinary means fail, an operation is indicated even when its magnitude appears to be out of all proportion to the purulent focus in the ear. In many cases the only certain means of preventing a threatened cerebral complication is to expose freely all the cavities of the tympanum, an operation which is quite devoid of danger if the operator is faultless in his technic and master of the anatomic relations.

Of all chronic diseases, cholesteatomata are the most dangerous. Though their progress is slow, they exert a constant, progressive, destructive influence, and nearly always lead to extensive destruction of the temporal bone and ultimate exposure of the interior of the cranium.

The meninges and sinuses are exposed to infection both by virtue of their direct contact with the diseased portions of the temporal bone and by the possibility of extension of a purulent otitis media to the dura through the fissures which exist in the bony plates separating the tympanum from the interior of the skull, especially in the roof of the tympanum. Or the infection may be carried through the labyrinth after the fenestræ have been destroyed by the sup-

purative process or the external wall of the labyrinth has become carious and pierced by fistulæ.

The mode of infection in all those cases in which the bone is found diseased up to the point where it comes in contact with the dura needs no explanation, but there are other cases of cerebral complications in which the bone was not found to be diseased up to that point. In explanation of such cases Körner has erected an osteophlebitis which, as will again be referred to, appears to be of special significance in the production of otitic pyemia. From this point of view the fistulæ which are often found in the bone running to an extradural abscess or to the diseased sinus represent periphlebitic blood-vessels.

The short review which we are about to give of the various forms of otitic cerebral disease is based on the assumption that the cerebral complication depends on the seat and variety of the aural disease, according to which the middle ear, the posterior cerebral fossa, the dura mater or pia mater will be affected. It also plays an important part in the localization of the secondary cerebral abscesses.

Diseases of the meninges in the middle fossa of the cerebrum corresponding to the temporal lobe are undoubtedly due to the passage of pus through the roof of a carious tympanum, but the importance of preformed openings in this plate of bone, which is naturally quite thin, has been greatly overestimated.

The transverse sinus becomes secondarily involved during its course within the sigmoid sinus as a consequence of the extension of caries of the mastoid antrum and cells to the posterior wall of the mastoid process. The danger of this complication depends on individual anatomic relations, for the course of the sinus varies with the general formation of the cranium, as was pointed out by Körner, it being more or less superficial and therefore nearer to, or farther removed from, the cells in the mastoid process. Thrombosis of a sinus is to be attributed to osteophlebitis even in those cases in which the caries has not reached the sinus (Körner), as the infectious thrombi in the smaller blood-vessels in the bone "grow into the sinus." Leutert [1] has introduced a new pathologic factor in isolated thrombus

[1] "Arch. f. Ohr.," vol. XLI.

formation, which he found in the bulb of the jugular vein and from which he deduced a retrograde thrombosis in the sinus. In thrombosis of a sinus there may be extension of the thrombus in the opposite direction of the blood current in the horizontal portion of the transverse sinus as far as the torcular Herophili, and as this leads to occlusion of the mastoid artery, there is a swelling over its point of exit behind the mastoid process which may be of great diagnostic value. The thrombosis spreads to the superior and inferior petrosal sinuses, and from them to the cavernous sinus, or it may extend downward into the jugular vein, but in that case it rarely extends beyond the mouth of the facial vein.

The dura of the posterior fossa of the cranium becomes diseased either after caries of the mastoid process or after suppuration in the labyrinth. The extension of mastoid disease to the dura of the posterior fossa depends on the arrangement of the system of cavities and the extent of the mastoid cells. As the cells are lined with epithelium, they permit the suppurative process to go on rapidly, and if they extend as far as the vitreous table, the process spreads more rapidly to the interior of the brain than when they are separated from the interior of the skull by a thicker layer of compact bone. It is to be remembered that there is a difference between adults and young children in this respect, as pointed out by Toynbee and later by Jansen.[1] As a result of the anatomic structure of the mastoid process, which in early infancy possesses but few cells arranged in a horizontal layer while the posterior wall which separates it from the petrous portion of the temporal bone is strongly developed, diseases in children up to the age of two years tend to invade the cerebrum and the middle, rather than the posterior, fossa.

Operations are frequently interrupted by the finding of an extradural abscess between the bone and the dura, the quantity of pus varying from a few drops to 15 c.c. From the expansive pulsation it is easy to recognize the endocranial origin of the discharge. The dura recedes before the pressure of the pus, and an abscess cavity is formed between the bone and the dura which leads to compression of the cerebral substance, and sometimes attains such

[1] "Arch. f. Ohr.," XXXV, p. 261.

enormous dimensions that the pus makes its way through the foramen magnum or the anterior jugular foramen into the deep muscles of the neck, or it may burrow along the lateral wall and base of the brain and make its way out directly through the bone, to form a deep abscess in the neck.

Extradural abscess is usually associated with an accumulation of pus in the external wall of the sinus, which exposes the latter to the danger of thrombosis. Although both the dura and the wall of the sinus may successfully resist this disintegration for a long time, the condition must eventually lead to infiltration and the formation of granulations, which sooner or later bring about the destruction of both structures. As, however, the course of the disease is very slow, adhesions frequently form between the dura and the pia mater and brain substance, thus preventing a purulent leptomeningitis.

Next to thrombosis of a sinus, cerebral abscess is the most frequent sequel of extradural abscess; but after the pus has been discharged through the diseased temporal bone and the extradural abscess has healed, the path of the otogenic infection is withdrawn from clinical observation and the interpretation of the abscess becomes difficult.

Suppurations in the labyrinth often lead to diseases of the dura of the cerebellum, because all preformed openings in that situation lead to the posterior fossa. Since the recent additions to our knowledge of the involvement of the labyrinth in purulent otitis media the significance of suppurative processes in the labyrinth in the production of otitic cerebral diseases is now better understood (compare Jansen and others [1]).

Once the pus has reached the labyrinth, it finds many channels through which it can enter the interior of the cranium, and we can readily understand that it is more likely to make its way in this direction than toward the middle ear, from which it is separated by the robust wall of the labyrinthine capsule. The channels referred to include the porus acusticus internus, the aqueduct of the vestibule and cochlea, which all have this in common, that they open on that surface of the petrous portion of the temporal bone which is directed toward the posterior fossa,

[1] "Arch. f. Ohr.," xxxv.

so that, as we have said above, they convey the pus to the meninges of the cerebellum.

Closely connected with the conception of otitic cerebral disease is that of *otitic pyemia*, which occurs in a great variety of forms, a distinction being drawn between otitic pyemia due to disease of a sinus and otitic pyemia of osteophlebitic origin without phlebitis of a sinus, and between these and an otitic septicemia.

If the above-mentioned theory of Leutert, that all cases of pyemia are due to the presence of thrombi, however minute, within the sinus or the bulb of the jugular vein is accepted, the matter is somewhat simplified, as the two first-mentioned forms of pyemia—that produced by thrombosis of a sinus and that produced by osteophlebitis—are united under one head. To reject the existence of an osteophlebitic pyemia for the sake of justifying this theory, which has never been perfectly proven, would be simply to ignore the observations of accurate observers.

It would lead me too far afield to go into the details of the similarity of these various forms of pyemia following diseases of the ear. In general, it may be said that in a sinus pyemia the most conspicuous feature of the clinical picture, next to the chills and abrupt rises in temperature, are the pulmonary metastases due to emboli from the disintegrating thrombus; while in osteophlebitic pyemia the fever is high, and shows neither typical chills nor constant remissions, emboli are less frequent, and when they do occur usually affect the joints and muscles, "as the microorganisms which get into the circulation in osteophlebitis are not incased in large portions of thrombic tissue, and are therefore easily able to pass through the lungs, until they are arrested somewhere in the capillary system of the systemic circulation" (Körner). Otogenic septicemia, finally, presents all the appearance of general sepsis such as we are accustomed to see in grave infections of the entire organism. If the habit of making a routine examination of the ear in all cases of septicemia could be formed, the practice would unquestionably result in a marked limitation of our present vague conception of cryptogenetic septicemia (septicopyemia).

INDEX.

Abscess, 333
cold, of the posterior pharyngeal wall in caries of the vertebræ, 162
extradural, in aural diseases, 334
tuberculous, of the thyroid cartilage, 154
Acid, salicylic, effects of, on the ears, 248
on the pharynx and larynx, 247
Acids, toxic effect of, on mucous membrane of pharynx and larynx, 242, 243
Acromegaly, manifestations of, in the nose, larynx, and pharynx, 95
Actinomycosis, laryngeal complications of, 186
of the mouth and pharynx, 185
Adenoid habit, 42
vegetations, 41
mouth-breathing in, 18
occlusion of the tubes and aural affections in, 36
reflex neuroses from, 313
Adhesions in the larynx from syphilitic ulcers, 218
in the pharynx, 213
Air-passages, upper, alterations of, in diseases of the lungs, 31
in diseases of the mediastinum, 33
in erysipelas, 147
in influenza, 132
in leukemia, 83
hemorrhages from, in arteriosclerosis, 52
in cardiac disease, 52
relation of, to ears, 35
scleroma of, 31
significance in respiration, 19
Alcohol, abuse of, effect on the organ of hearing, 250
on the structure of the pharynx, 249
Alkalies, destruction of the pharyngeal and laryngeal mucous membrane in poisoning by, 242
Analgesia of the mucous membranes of the upper air-passages in hysteria, 297
Anemia, general, appearances in the ears, 82
in the nose, pharynx, and larynx, 81
of the laryngeal mucous membrane in tuberculosis, 168
Anesthesia dolorosa, 298
in hysteria, 297
of the nose, throat, and larynx after diphtheria, 142
in epilepsy, 294
in progressive amyotrophic bulbar paralysis, 292
significance of, in inspiration pneumonia, 22
Aneurysm of the aorta, motor disturbances of the larynx, 55
pressure-symptoms in the trachea, 54
of the large blood-vessels, subjective noises in, 54
Angina, catarrhal, in acute rheumatoid arthritis, 140
in measles, 108
in scarlatina, 112
edematous, after the use of salol, 247
uratica, 102
Anthrax, infection of the nose and pharynx in, 185
Antimony poisoning, hoarseness in, 247
Antipyrin and antifebrin, olfactory disturbances after use of, 247
tinnitus aurium and difficult hearing after use of, 248
Aphasia in diabetic hemiplegia, 96
uremic, 189
Aphonia in arsenic eaters, 245
in Asiatic cholera, 75
in chlorosis, 81

Aphonia in hysteria, 304
in malaria, 148
Aprosexia in mouth-breathers, 312
varieties and pathogenesis, 312
Arsenic-poisoning, acute and chronic ulcers in the external auditory meatus in, 245
nasal and pharyngeal catarrh in, 245
Arteriosclerosis, hemorrhages in the upper air-passages, 52
tinnitus aurium, 53
Arthritis, gonorrheal, of the articulations of the larynx, 204
Asthenopia in hypertrophy of the turbinates, 230
Asthma in relation to the nose, 306, 307
to the sexual functions, 199
uremic, differential diagnosis from bronchial asthma and laryngeal stenosis, 188
Astringents, injurious effect of, on the sense of smell, 250
Atelectasis as a cause of middle-ear disease, 50
Auditory disturbances during menstruation and pregnancy, 202
from increased intracranial pressure, 268
in anemia, 81
in aural tuberculosis, 174
in cerebellar disease, 273
in disease of the central nervous system, 263, 271, 272
in disease of the meninges, 326
in epilepsy, 294
in facial paralysis, 323
in hysteria, 300
in intoxications, 242
in leukemia, 86
in multiple sclerosis, 290, 291
in nephritis, 189
in parotitis epidemica, 138
in pseudoleukemia, 89
in syphilis, 220
in tabes dorsalis, 278
in typhoid fever, 132
nerve, atrophy of, in amyotrophic bulbar paralysis, 292
in tabes, 283, 286, 288
hemorrhage and lymphatic infiltration in leukemia, 84
neuritis of, from abuse of tobacco, 250
paralysis of, in hereditary syphilis, 223
pathways in the central nervous system, 271
Auditory nerve, reactions of, electric, diagnostic significance of, 265
increased irritability of, 269
in hysteria, 301
in rabies, 186
in strychnin-poisoning, 247
methods of examination of, 266
normal formula, 265
inversion of, 270
paradoxic, 269, 270
reflex effect on the movements of the eye, 237, 238

Bacillus mucosus ozœnæ of Abel, 46
Blennorrhea neonatorum as a complication in diseases of the nasal mucous membrane, 226
Blepharospasm from spasm of the stapedius muscle, 240
in atrophy of the nasal mucous membrane and adhesions within the nose, 230
Blood, diseases of, pathologic appearances in the upper air-passages, 81
Bronchial glands, diseases of, and effect on respiration, 34
Bronchiectasis as a cause of middle-ear disease, 50
in disease of the upper air-passages, 28
Bronchitis after purulent otitis media, 51
capillary, as a cause of aural diseases, 50
chronic, in scrofulous children, 29
fetid, as a sequel of suppuration in the accessory cavities of the nose, 28
laryngitis and pharyngitis after, 31
significance of mouth-breathing in production of, 29
Bronchopneumonia in connection with purulent otitis media, 50, 51
Bruits laryngés in hysteria, 303
Bulbar palsy, progressive amyotrophic, laryngeal and aural symptoms in, 293

Caries of the accessory cavities of the nose, etiologic significance in diseases of the meninges, 330
of the temporal bone in tuberculosis, 173
sicca of the nose in syphilis, 208

Central nervous system, diseases of, as the cause of auditory disturbances, 263
of laryngeal affections, 251
Cerebral disease, otitic, 334
sinuses, disease of, 325, 328, 330, 332
Cerebrospinal meningitis, acute, significance of the nose in, 326
of the ear, 327
deafness in, 327, 328
in combination with purulent otitis media, 327
Chlorid of zinc poisoning, appearance of the pharyngeal and laryngeal mucous membrane in, 243
Chloroform narcosis, effect of, on the organ of hearing, 249
Chlorosis, aphonia in, 81
Choked disc in chronic middle-ear disease, 238, 239
Cholera Asiatica, effect of, on upper air-passages, 75
Cholesteatoma as cause of cerebral disease, 330, 331
Chondritis laryngea in typhoid fever, 127
primary tuberculous, 153
Chorda tympani, lesions of, in relation to sense of touch, 320
origin and course of, 321
Chorea minor, motor disturbances of the aural and laryngeal muscles in, 295
Choreic movements in nasal obstruction, 313
Chronic acid-poisoning, chronic disease of the upper air-passages from, 243
Circulatory system, diseases of, in relation to the upper air-passages, 50
Cirrhosis of liver, hemorrhages in, 74
Cohabitation in relation to the nose, 197
Condylomata in the auditory meatus in syphilis, 219
Conjunctivitis in scrofulous rhinitis, 228
Constitutional diseases, chronic, pathologic alterations of the upper air-passages in, 92
Coordination, disturbances of, in hysteria, 301
of the laryngeal muscles, disturbances of, in chorea minor, 295
Coryza neonatorum in hereditary syphilis, 210
Coryza, vasomotor, 306, 308. (See also *Hydrorrhea nasalis*.)
in malaria, 148
Cough due to gall-stones, 75
in nasal affections, 307
nasal, 306
trifacial, 307
Croup, pharyngeal and laryngeal, in cholera, 75

Dacryocystoblennorrhea after disease of the nose, 226
Deafness after embolism of the internal auditory artery, prognosis of, 67
during menstruation, 203
in alcohol and tobacco intoxication, 249
in brain-lesions, 271
in cerebrospinal meningitis, 328
in chloroform narcosis, 249
in combination with subjective noises, 64
in diseases of the meninges, 326
in epilepsy, 294
in facial paralysis, 323
in hysteria, 300
in lead-poisoning, 246
in leukemia, 87
in multiple sclerosis, 203
in nephritis, 189
in potassium iodid poisoning, 244
in quinin and salicylic acid poisoning, 248
in syphilis, 221
in tabes dorsalis, 280
in typhus and typhoid, 131
postdiphtheric, 146
Deformities, nasal, in syphilis, 209
Diabetes mellitus, aural complications in, 97
dryness and atrophy of the oral mucous membrane in, 96
ictus laryngis in, 105
mastoid disease in, 98
Digestive organs, diseases of, in relation to nose, throat, and larynx, 70
Diphtheria, deafness in, 146
diseases of the ear in, 143
in cholera Asiatica, 75
in measles, 109
in scarlatina, 113
in typhoid, 126
postdiphtheric palsies in pharynx and larynx in, 142
Diplococcus pneumoniæ in purulent otitis media, 49

Dyspepsia as a cause of rhinopharyngeal disease, 73
due to disease of the nose and pharynx, 72
Dyspnea in laryngitis acutica rheumatica, 141

Ear, disease of, in connection with mastication and deglutition, 77
in diphtheria, 143–145
in infants, 78, 79
in influenza, 134
in leukemia, 86
in malaria, 149
tubercular, 169
effect of various diseases of the respiratory organs on, 47
embolic disease of, in endocarditis, 67
innervation of, 316
in relation to diseases of the heart and blood-vessels, 59
relation of, to upper air-passages, 35
vascular systems of, 60
Earache, 47. (See also *Otalgia*.)
in diseases of the respiratory organs, 47, 48
Eczema of the nose, 159
etiologic significance of, in erysipelas, 147
in scrofulous children, 160, 193
Edema of the upper air-passages in nephritis, 188
aural disturbances in, 190
Emboli in the ear during the puerperium, 203
Emphysema, relation of, to chronic catarrh of the upper air-passages, 28–30
Empyema of antrum of Highmore, 70
Endocarditis, embolic disease of the organ of hearing in, 67
Enteritis in children in connection with inflammation and suppuration of the middle ear, 78
Entotic vascular murmurs, character of, 61, 62, 65
differential diagnosis from simple noises in the ear, 66
Enuresis nocturna, etiology of, 313
Epiglottis, cicatricial changes in syphilis, 219
function and importance of, 28
in acute rheumatic laryngitis, 140
in lupus, 175
Epiglottis in typhoid, 127
leprosy of, 182
otalgia in inflammatory swelling of, 47
ulcerations of, in foot-and-mouth disease, 184
Epilepsy, symptoms of, in the larynx and in the organs of hearing and taste, 294
Epileptic equivalent in nasal obstruction, 314
Epiphora from aural irritation, 240
from nasal irritation, 240
in obstruction of the lacrimonasal duct, 226
Epistaxis in arteriosclerosis, 52
in cardiac disease, 52
in cirrhosis of liver, 74
in hemophilia, 89
in leukemia, 83
in malaria, 148
in purpura hæmorrhagica, 90
in scorbutus, 91
in typhoid fever, 124
seat of, 53
Erysipelas of the upper air-passages, 147
secondary to eczema of nose and ears, 148
Erysipèle cataméniale, phenomena and reflex character of, 199
Erythema exsudativum multiforme, 196
of the larynx, 215
of the nose, 205, 206
of the upper air-passages, 193
syphilitic, of the gums and tonsils, 210
Esophagus, diseases of, effect on upper air-passages, 72
Eunuch voice, etiology and cure of, 201
Eustachian tube, dilatation of, in atrophic rhinitis, 45
functions of, disturbances of, 36
due to alterations in upper air-passages, 40
occlusion of, 38
as cause of middle-ear disease, 42
by atrophy of pad of fat, 46
due to paralysis of muscles of soft palate, 43
in acute and chronic catarrh of upper air-passages, 41
in relation to nasal stenosis, 42
significance of, in middle ear, 37

Exanthemata, acute otitis media in, 44, 109, 116, 119
manifestations of, in upper air-passages in measles, 108
in scarlet fever, 112, 113
in varicella and variola, 121
Exostoses in the external auditory meatus in acromegaly, 96
External auditory meatus, diphtheria of, 143
exostosis and hyperostosis of, in acromegaly, 96
furunculosis of, in diabetes, 96
hyperesthesia and hyperalgesia of, in hysteria, 298
in gout, 103
in syphilis, 220
pressure variations in, 45
respiratory murmurs in, 45
scarlatinal diphtheria of, 118
skin diseases of, 196
stenosis of, from syphilitic scars, 220
syphilitic affections of, 219
Eyes, diseases of, in pathologic conditions of the nose and its accessory cavities, 225
in alterations in the ear, 235
reflex disturbances of, movements of, from the ear, 237
in acute and chronic otitis media, 239

Facial paralysis, 324
causes of, 324
course of, 325
in middle-ear disease, 325
localization of lesion in, 323
Fish-poisoning, appearance of upper air-passages in, 248
Foot-and-mouth disease, catarrh of the tubes in, 185
infection through mucous membranes of upper air-passages, 184
Frontal sinus, neuralgia in, 314
suppuration of, hyperemia and venous stasis of the optic papilla of the same side in, 233

Ganglion, Gasserian, in purulent otitis media, 320
sphenopalatine, disease of, in caries of the sphenoid and ethmoid bones, 316
Gasserian ganglion, effects of extirpation of, 318
Gastro-intestinal disease as cause of middle-ear disease, 78
Gingivitis in leukemia, 84
Glanders, infection through mucous membranes of upper air-passages, 183
Glottis, spasm of, 306
hysteric, 307
Gonorrhea, infection of the nose by secretions of, 203
laryngeal disease in, 204
Gout, angina uratica in, 102
aural manifestations in, 103
changes in the ear in, 102, 104
in the larynx in, 102, 104
in the pharynx in, 101
ictus laryngis in, 105
Gumma in the external ear and mastoid process, 220
in the larynx, 215, 217
in the nose, 206
in the pharynx, 211, 212
Gums, affections of, in leprosy, 181
in leukemia, 85
in lupus, 176
in measles, 108
in middle-ear disease, 43
in typhoid fever, 124
syphilitic, 210, 211
ulcerative, in foot-and-mouth disease, 184

Hallucinations, auditory, in abuse of alcohol and tobacco, 250
in chloroform narcosis, 249
in epilepsy, 294
Hay-fever, 306, 308
Heart and blood-vessels, diseases of, in relation to the nose, pharynx, and larynx, 52
to the ear, 59
lesion, hemorrhages in mucous membranes in, 37
tinnitus aurium in, 60
Hemophilia, hemorrhages in upper air-passages and ears in, 88, 89
Hemorrhages from the ears in hysteria, 304
in vicarious menstruation, 201
in affections of the genitalia, 197
in cirrhosis of liver, 74
in hemorrhagic diathesis, 89
in leukemia, 83, 84, 87, 88
in pseudoleukemia, 85
in the middle ear in nephritis, 189

Hemorrhages in the mucous membranes of the upper air-passages, 53
Hemorrhagic diathesis, 89
Herpes in ear and external meatus, 196
on mucous membranes, 194
Hoarseness from abuse of alcohol and tobacco, 249
in poisoning with antimony, copper, and phosphorus, 247
in syphilis, 213
Hydrorrhea nasalis in hepatic colic, 75
in malaria, 148
Hyperacousis, 324
Hysteria, disturbances in the nerves of special sense in, 300
in the ear in, 298
muscles of, 304
motor, in larynx, 301
sensory, in the nose and pharynx, 297
Hysterogenetic zones in the mucous membranes of the upper air-passages, 298

Icterus in otitis media, 80
Ictus laryngis in obesity, gout, and diabetes, 104
Influenza, 132
aural complications in, 134
laryngitis hæmorrhagica, 134
nasal complications in, 133
palsies in, 134
purulent otitis media in, 137
Inspiration pneumonia, causes of, 27
Internal ear in influenza, 137
Intoxications, appearances of, in the upper air-passages, 241
auditory disturbances in, 242

Jaundice in mucous membranes, 74

Keratitis, eczematous, in eczema of the nose in scrofulous children, 227
Kidneys, diseases of, 188. (See also *Nephritis.*)

Labyrinth, diseases of, 333
Lacrimonasal duct as a carrier of infection from the nose to the eye, 225
Laryngeal crises in tabes dorsalis, 278
Laryngismus stridulus, 93
Laryngitis acuta rheumatica circumscripta, 141
hæmorrhagica, 134
leukemic, 84
Laryngospasm. (See *Larynx.*)
Larynx as respiratory pathway, 25
edema of, after use of potassium iodid, 244
in influenza, 133
in malarial cachexia, 148
in nephritis, 188
in typhoid, 124
extirpation of, followed by death from heart failure, 45
in relation to diseases of heart and blood-vessels, 52
to nose and pharynx, 18
paralysis after disease of the lungs, 32
from struma, 35
in cardiac and vascular disease, 53
in tumors of mediastinum, 34
relation of, to diseases of digestive organs, 70
spasm of, 92
tuberculosis of, 153–158
ulcers of, anatomic and clinical varieties of, 125
in croupous pneumonia, 31
in leprosy, 182
in lupus, 176
in typhus and typhoid, 123
pathogenesis of, 126
tuberculous, 156
Lateral sclerosis, amyotrophic laryngeal paralysis in, 293
Lead poisoning, chronic, manifestations of, in the ear, 246
paralysis of the laryngeal muscles in, 246
Leprosy, early appearances in, 179
of the ear, 183
of the nasal cavities, 177
clinical picture, 180
localization of nodes and ulcers in, 181
pathogenesis, 178
of the pharynx, 181
transmission of, by nasal secretion, 179
Leukemia, 83
alterations in upper air-passages in, 83
hemorrhages in, 83
manifestations of, in the ear, 86

Localization of aural disturbances, 326
of the ear in the brain, 270
of the movements of the vocal cords, 258
Locus Kieselbachii, 53
Lungs, diseases of, due to disturbances in the upper air-passages, 26
in morbid conditions of upper air-passages, 28
in relation to nose, 19
Lupus in the upper air-passages, 175
localization of, 176
morbid anatomy of, 175
of the external ear, 177
of the eye, 228
of the pharynx, 176
sequels of, 176
Lymphomata in pseudoleukemia, 85, 86
leukemic, of the internal ear, 87
of the pharyngeal structures, 84
Lymphosarcoma, 85
of the pharyngeal structures, 86

Malaria, 148
aphonia in, 149
epistaxis in, 148
hydrorrhea nasalis in, 148
vasomotor rhinitis in, 148
Malarial disease of the ears, 149
Mastoid disease as cause of cerebral diseases, 333
extension of, to dura, 333
in diabetes, 97–99
syphilitic, 219
tuberculous, 171, 174
Masturbation, effect of, on existing aural disease, 202
epistaxis in, 197
tinnitus aurium in, 202
Measles, 109
aural complications in, 109–111
croupous laryngitis in, 109
Koplick's sign in, 109
Mediastinum, diseases of, effect on respiration, 33
Medulla oblongata, diseases of, motor and sensory disturbances in the larynx, ear in, 291
Menière's symptom-complex in tabes, 280, 283
pathogenesis of, 263
relation of, to gout, 104
to mumps, 137
Meninges, diseases of, in connection with cranial nerves, 325
in nasal affections, 328
Meninges in relation to aural affections, 330
Meningitis, purulent, as cause of paralysis of cranial nerve, 326
in relation to nose, 329
paralysis of olfactory disturbances in, 326
of vocal cords in, 326
Menstruation, relation of, to nasal affections, 197
Mercurial poisoning, appearances of, in mouth and pharynx, 246
in sound-perception apparatus, 246
Middle ear, bacteria in, 44
catarrh of, acute and chronic, 38
changes in pressure in, 37
disease after atelectasis, 50
after bronchiectasis, 50
after capillary bronchitis, 50
diabetic otitis, 99, 100
in leukemia, 88
in measles, 110
in scarlatina, 118
in typhoid fever, 129
diseases of, due to infection from postnasal space, 43
due to obstruction of Eustachian tube, 41
hydrops ex vacuo in, 38
hyperæmia ex vacuo in, 38
icterus in otitis media, 80
inflammatory and noninflammatory catarrh of, 41
otitis media sclerotica, 45
purulent otitis media as cause of bronchitis and bronchopneumonia, 51
as sequel of gastro-intestinal disease, 79
in cerebrospinal meningitis, 327
in connection with caries of the teeth, 77
with dentition, 77
in influenza, 137
in pneumonia, 48
therapeutic infection of, through Eustachian tube, 46
variations of pressure in, 39
Miliary tuberculosis, acute, beginning in the larynx and pharynx, 154
Mountain sickness, 40
Mouth, affections of, gonorrheal, 204
in actinomycosis, 185
in foot-and-mouth disease, 184
in leukemia, 85
in mercurial poisoning, 291
in mouth-breathers, 68

Mouth affections in varicella, 121
Mouth-breathing as a cause of chronic bronchitis, 29
causes of, 26
effects of, 26
Muscular atrophy, progressive spinal, laryngeal palsy in, 293

Nasal douches, dangers of, 46
proper use of, 47
Nasalis luetica, 208
Nephritis, complications of, aural, 190
in the upper air-passages, 188
Nervous diseases of the larynx, 251
of the organ of hearing, 263
with changes in the nose, throat, and larynx, 275
Neuralgia, infra-orbital, 315
otitica, 317
supra-orbital, 314
trifacial, 314, 315
in relation to ear, 318
in sarcoma of the ear, 318
of Gasserian ganglion, 318
tympanica, 317
Neuritis, gonorrheal, of the larynx, 205
optica in papillomata of the turbinates, 231
Neuroses, involvement of the nose, throat, larynx, and ears in, 293
Nose as respiratory pathway, 20
bacteria in, 22, 44
bactericidal power of secretion, 23, 44
hydrorrhea of, 308
innervation of, 306
in relation to diseases of digestive organs, 70
of the heart and blood-vessels, 52
to lungs, 19
lupus of, 176
reflex irritation of, from intestines, 74
neuroses of, 306
sneezing reflex, cough, etc., 306
stenosis of, in relation to occlusion of Eustachian tube, 42
tuberculosis of, 153, 158–161
eczema in, 159

Obesity, ictus laryngis in, 104
Olfactory nerve, hyperesthesia of, in epilepsy, 294
Olfactory nerve, in hysteria, 297
in rabies, 186
reflex effects of, 308
sense, disturbances of, in anemia, 81
function of, in respiration, 24
Oral cavity, diseases and changes in form of, in disturbances of nasal respiration, 68
Ossicles of the ear, caries of, in tuberculosis, 174
rheumatic disease of, 141
Osteitis luetica of the nose, 207
Osteophlebitis as cause of otitic pyemia, 332
Otalgia, 317
in caries of the teeth, 76
in diseases of the respiratory organs, 47
of the trifacial, 317
in hysteria, 299
tympanica in influenza, 137
Otitis media catarrhalis, 39
neuroparalytica, 319
Oxyocoia, 324
Ozena, 29
pathogenesis of, 30
relation of, to leprosy, 177
to menstruation, 199
Ozœna syphilitica, 208

Palate, paralysis of, in influenza, 133, 134
in progressive amyotrophic bulbar paralysis, 292
occlusion of tube in, 43
postdiphtheric, 142, 143
unilateral, in typhoid, 128
Papillitis nervi optici in purulent otitis media, 239
Papules, syphilitic, in the auditory meatus, 219
in the larynx, 215
on the gums, 210
on the nasal mucous membrane, 206
on the tonsils, 210
on the tympanic membrane, 219
Paralysie glosso-labio-laryngée, 292
Paralysis agitans, motor disturbances of the vocal cords in, 293
of the recurrent, definition, 54
from pericardial exudate, 58
in aneurysm of aorta, 55, 56
postdiphtheric, 142
Parasites, intestinal, reflex symptoms of, in the nasal mucous membrane, 74

Paresis of the vocal cords in laryngeal tuberculosis, 168
Paresthesia of the upper air-passages
from sexual excitement, 200
in amyotrophic bulbar paralysis, 292
in chlorosis, 81
in hysteria, 298
Parosmia from sexual excitement, 200
in hysteria, 300
in influenza, 134
Parotitis epidemica, 138
Pericardial exudate, paralysis of recurrent form, 58
Perichondritis in typhoid, 134
laryngeal, in influenza, 134
nasalis luetica, 207
syphilitica, 215
tuberculous, primary, 154
secondary, 166–168
ulcerative, 125
Pertussis, 311
Pharyngeal crises in tabes dorsalis, 279
Pharyngitis, leukemic, 84
Pharynx as respiratory pathway, 25
edema of, in nephritis, 188
in relation to diseases of the digestive organs, 70
of the heart and blood-vessels, 52
to larynx, 19
to nose, 18
lupus of, 176
retropharyngeal abscess of, 162
Phenomena, irritative, in diseases of the meninges, 325
motor, of the laryngeal muscles, 251
sensory, in the eyes from irritation in the ears, 239
Phonation, disturbances of, in hysteria, 302
Pneumonia, laryngeal complications in, 32
purulent otitis media in, 48
Politzer's method of inflating tympanum, 46
Posticus paralysis, 256
in gonorrheal arthritis, 205
in hysteria, 302
in syphilis, 219
in tabes dorsalis, 275
median position of vocal cord in, 254, 255
Pregnancy, aural affections in, 202
nasal affections in, 197
Pseudobulbar paralysis, laryngeal paralysis in, 293
Pseudoleukemia, 85
auditory disturbances in, 89
Puerperium, aural affections in, 202
Pupil, changes of, from nasal irritation, 231
in purulent otitis media, 239
Purpura, 89
Pyemia, otitic, 332–335

QUININ, toxic effect of, on the organ of hearing, 248

RABIES, implication of the auditory sphere in, 186
nervous symptoms in, 186
Rachitis, aural disease in, 95
laryngeal spasm in, 93
Raucego syphilitica, 213
Recurrent paralysis, 252
cadaveric position of vocal cords in, 253, 254, 258
diagnostic significance of, 252
from pericardial exudate, 58
in aneurysm of aorta, 54
in arsenic-poisoning, 245
in influenza, 135
in leukemia (tumors), 85
in mediastinal tumors, 33
in pulmonary tuberculosis, 33
in swelling of bronchial glands, 34
in tabes dorsalis, 276
laryngoscopic image in, 253
stage of posticus paralysis in, 254
Reflex neuroses. laryngeal, 106
nasal, 59, 305
palpitation of the heart due to irritation in the nose, 59
pathogenesis of, 309
Reflexes of sexual apparatus, 308
olfactory, 309
Refraction, errors of, due to nasal hypertrophies, 231
Respiration, changes of pressure in the external auditory meatus in, 37
nasal, effect of disturbances of, on oral cavity, 68
physiologic pathway of, 20
preparation of the inspired air, 20
reflex influence of the olfactory on, 309
significance of upper air-passages in, 19
Respiratory air current, removal of foreign substances and microorganisms from, 21

Respiratory air current, warming and saturation of, 23
murmur, inspiratory and expiratory, in the ear, 37
Retina, peripapillary opacity of, in suppuration of the frontal sinuses, 233
Rhinitis, acute, in influenza, 132
atrophic syphilitic, 208
diseases of the eye in, 226
fœtida atrophica, 29
hypertrophic syphilitic, 207
purulent, from infection with gonococci, 203

SADDLE-NOSE, characteristics and mode of production of, 209
Saliva, secretion of, in disease of chorda tympani, 323
Salivation, effect of irritation of the tympanic plexus and chorda tympani on, 322
Salol, edematous angina after the use of, 247
Sausage poisoning, dryness of the throat and hoarseness in, 248
Scarlatina, 112
acute otitis media in, 118
aural complications in, 115
diphtheroid, 113
Scleroma of upper air-passages, 31
Scorbutus, 89
Sensory disturbances of the eyes from irritation in the ear, 239
hysterical, 296
Septicemia, otogenic, 335
Septum, nasal perforation of, in glanders, 184
in leprosy, 177
in lupus, 176
in tuberculosis, 159
Sexual functions, reflex effect of, on upper air-passages and ears, 197
Silver-nitrate poisoning, pigmentation of the mucous membrane of the mouth, tongue, larynx, and tympanic membrane in, 247
Skin, diseases of, complications of, in the external ear and meatus, 196
in the upper air-passages, 193
Sneezing reflex in respiration, 24, 306, 307
Speech disturbances in chorea minor, 295
in paralysis agitans, 293
Spinal cord, diseases of, pathologic changes in the nose, throat, larynx, and ears in, 275
Stapedius muscle, effect of paralysis of, 323, 324
Stomatitis. (See *Mouth, Affections of.*)
aphthosa epidemica, 184
in leukemia, 85
Strabismus as a complication of hypertrophied tonsils, 230
Stridor, inspiratory, from enlarged thymus, 35
Struma as cause of tracheal stenosis, 34
Sympathetic nerve, paralysis of, etiologic significance in hydrorrhœa nasalis, 306
Syphilis, nervous deafness in, 221, 222
of the organ of hearing, 219
in hereditary lues, 222
of the upper air-passages, 205
primary lesion of, in the nose, 206
in the pharynx, 210
secondary manifestations, of in the larynx, 215, 216
in the nasal mucous membrane, 206
in the pharynx, 210
tertiary manifestations of, in the larynx, 215, 216
in the nose, 206
in the pharynx, 211
Syringomyelia, motor and reflex disturbances in the larynx and posterior wall of pharynx in, 291

TABES dorsalis, involvement of auditory nerve in, 279
of the laryngeal nerves in, 275
of the olfactory nerve in, 275
Taste, disturbances of, in middle-ear disease, 321
Teeth, diseases of, in relation to antrum of Highmore, 70
to nasal cavities, 71
Tensor tympani, disturbances of, 318
Thrombosis of cerebral sinus, 332, 333
Thymus gland, alterations of, effect on respiration, 33, 35
as cause of inspiratory stridor, 35
as cause of sudden death in children, 35
Tic convulsif due to nasal obstruction, 313

Tinnitus aurium after wounds of the head, 62
caused by pressure on internal jugular vein, 67
in anemia, 62, 81
and hyperemia within the ear, 59
in aneurysm, 62
in arteriosclerosis, 62, 63
in disease of heart and blood-vessels, 59
in facial paralysis, 324
in leukemia, 87
in sclerosis, 62
in treatment of heart and lung diseases by rarefied and compressed air, 40
in variations of atmospheric pressure, 40
Tobacco, abuse of, auditory disturbances from, 250
pharyngeal catarrh from, 249
Tonsils, syphilitic disease of, 210, 211
Toothache distinguished from otalgia, 317
Trachea, changes in, produced by aneurysm of the aorta, 57
pressure ulcers and perforations of, 57
pulsating movements of, 57
rupture into, in aneurysms of aorta, 52
stenosis of, 57
from mediastinal tumors, 34
from struma, 34
in aneurysm of aorta, 52
Transfert of sensory disturbances in the ear in hysteria, 299
Trichinosis, paralysis of laryngeal and pharyngeal muscles in, 187
pharyngeal and laryngeal palsies in, 187
Trifacial, disease of, in relation to the ear, 316, 320
significance of, in rhinology and otology, 314
Tuberculosis, 151
of the ear, 169
chronic, 171, 172
diagnostic significance of bacilli in, 170
of the larynx, 153–158, 162–169
of the nose, 153, 158–161
of the pharynx, 153, 161
of the upper air-passages, 151
mode of infection in primary, 151
in secondary, 154
morbid anatomy of, 151
Tuberculosis, tonsillar, 152
Tympanic membrane, respiratory movements in, 45
retraction of, 38
signs and symptoms of, 38
rupture of, 39
sensory disturbances of, in hysteria, 298
syphilitic papules on, 219
Typhoid fever, 123
auditory disturbances in, 132
aural complications in, 128
changes in the cartilages of the larynx in, 127
complicated with diphtheria in, 126
epistaxis in, 124
mastoid disease in, 130
middle-ear diseases in, 129
palsies in, 127, 128
pharyngeal and laryngeal catarrh in, 124
ulcers in, 125

Ulcers, arrosion, tuberculous, of the larynx, 156
localization and pathology of, 164
tubercular, in the larynx, 164
in the nose, 160
in the pharynx, 151
Uremia, chronic deafness in, 191

Valsalva's experiment, 37, 46
Varicella, 120
Variola, 121
aural complications in, 122
palsies in, 122
Vasomotor disturbances of nasal origin, 307
varieties of, 308
Vertigo in anemia, 81
in leukemia, 87
laryngeal, 105
Visual disturbances after operative interference in the nose, 231
Vocal cords, laryngospastic attacks of, 55
movements of, choreic, 295
localization of, 258
paralysis of, in disease of the liver, 74
paresis of, in tuberculosis of, 168
periodic palsies of, 55

Vocal cords, spasm of, due to seat-worms, 74
tuberculous polyps of, 169
tumors of, in multiple sclerosis, 290
in paralysis agitans, 294
ulcerations of, in croupous pneumonia, 31
in leprosy, 182
in tuberculosis, 164, 165
Voice, change of, hyperemia of vocal cord in, 200
pathologic varieties of, 200
relation of, to puberty, 199
Vox cho'erica, 75

Xerosis of the mucous membranes, 29, 31, 45

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